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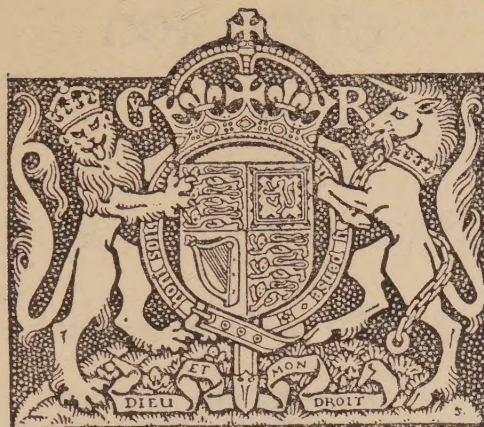
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# MANUAL OF TREATMENT OF GAS CASUALTIES

1930

WAR OFFICE

*By Command of the Army Council,*

*H. J. Creedy*

THE WAR OFFICE,  
4th July, 1930.

LONDON:

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# MANUAL OF TREATMENT OF GAS CASUALTIES

## FOREWORD

SUCCESS in minimizing the incidence of chemical warfare casualties, and in handling and treating such casualties, is bound to be proportionate to the extent to which information concerning the use of poisonous substances which may be adopted by the enemy for offensive purposes in warfare, the nature of the injuries caused by them and the lessons learnt during the Great War are the common knowledge of all medical officers.

Very complete information on these subjects is to be found in the Official History of the War, Medical Services, Diseases of the War, Vol. II. (H.M. Stationery Office, 1923).

As it is likely that in the event of future wars a considerable number of temporary medical officers will be employed with the Forces, the nature of whose civil occupation will have precluded their taking any particular interest in a subject which is of such great importance from a military point of view, whilst officers of the Royal Army Medical Corps will require to refresh their memories of it, it has been considered advisable to issue this manual, in order to give, in a portable form, those particulars which should be of practical value in the field.

It is certain that if an enemy resorts to this form of attack he will endeavour to circumvent our protective measures by using known substances more effectively, and in higher concentrations, over more extended areas, as well as by the introduction of new poisonous gases. It is of paramount importance that commanders should receive the earliest possible information of the use of gas in the field, and immediate report should, therefore, be made to the superior medical authorities on the appearance of symptoms amongst casualties indicative of poisoning by warfare gases. Special care should be taken to



note symptoms in any way different from those described in this manual, in order that steps may be taken without delay to afford adequate protection, and that medical officers in areas which have not experienced the new poison may have timely warning of the possibility of its use, together with any information which may help in the prevention of casualties and in the treatment of those affected. (Attention of Medical Officers is also directed to Defence against Gas, 1927, para. 16, page 17.)

As fresh information is circulated it must be inserted in the Manual. It is the duty of every officer to whom it is issued to ensure that this is carried out.

In addition to the more purely medical aspects of chemical warfare which are dealt with in the succeeding chapters, medical officers should realize that they are responsible for their own protection and for that of the personnel of their units, and of the patients under their care. They should, therefore, study Defence against Gas, to which this is a companion volume, and take advantage of every opportunity of familiarizing themselves with the use of defensive appliances. They should also accustom themselves to the use of respirators so as to be able to carry out their duties whilst wearing them without loss of efficiency.



## CHAPTER I

### NATURE OF ANTI-GAS DEFENCE

#### 1. Definition of Gas.

- (a) The term Gas, in connection with warfare, is very loosely used, but includes any chemical substance whether a solid, a liquid, or a gas employed for its poisonous or irritant effect on the human body. Such substances are, generally speaking, dispersed in the air as vapours or poisonous smokes, and exercise their action on personnel exposed to the contaminated air. Some of them, such as mustard gas, also act by direct contact of the liquid with the human body.
- (b) From a tactical point of view gases are generally divided into two main categories :—
  - Non-persistent,
  - Persistent.
- (c) Non-persistent substances when liberated are rapidly converted into gas or smoke ; clouds of gas so produced continue to be effective until dissipated by the wind and the sun.
- (d) Persistent substances used in gas warfare are generally liquids, which contaminate the area on which they are released and continue to give off vapour for a considerable period. Mustard gas and most tear gases are typical examples. Whilst evaporation is going on the immediate neighbourhood to leeward of the contamination is dangerous. In the case of gases such as mustard gas, which attack the skin, actual contact with contaminated ground or objects must be avoided.

#### 2. Factors Governing the use of Gas in the Field.—

The more important factors influencing the use of gas in the field are as follows :—

##### (A) *Meteorological Factors.*—

(1) Wind.—The lower the wind velocity the greater will be the effect of all gases. Strong winds rapidly disperse non-persistent gases. Persistent gases are affected in a slightly different manner. Over an area sprayed with mustard gas, for example, a strong wind would increase the rate of evaporation of the mustard gas, thus tending to clear the area more rapidly. The more rapid evaporation tends to produce a higher gas concentration ; this tendency is, however, more



than balanced by the increased dispersion. Absence of wind results in the formation of high local concentrations.

(2) Temperature.—A moderate air temperature is generally more favourable than either a high or low one, since it assists vaporization without seriously affecting persistence. Some substances, such as mustard gas, which are rightly classed as persistent at moderate temperatures, become solid at low temperature, and practically non-persistent at high temperatures, owing to increased rate of vaporization. Quite apart from the actual temperature, the variation of temperature with height above the ground is very important. If the temperature increases with height above the ground, then there is practically no convection in the atmosphere and consequently very little diffusion of gas upwards. If the temperature decreases with height above the ground, then the conditions are favourable for convection currents and the gas will diffuse upwards much more rapidly.

(3) Sunshine.—In bright, warm, sunny weather the temperature generally decreases with height above the ground. Thus, as explained in the last paragraph, upward air currents are formed which rapidly disperse gas and smoke clouds.

(4) Moisture.—Some screening clouds are greatly dependent on the amount of moisture in the air, a fairly high humidity being necessary to produce opacity. Heavy rain, however, is a disadvantage since it tends to wash the gas out of the air, and will also slowly destroy any liquid on the ground.

The most favourable conditions for the employment of gas are :—

- (a) Low wind velocity.
- (b) Temperature increasing with height above ground.
- (c) Moderate temperature.
- (d) Absence of heavy rain.

In the case of some screening clouds there must be added :—

- (e) Fairly high humidity.

These conditions frequently exist at the following times :—

- (a) Clear night. Wind 0—8 m.p.h.
- (b) Overcast night. Wind 0—4 m.p.h.
- (c) Overcast winter day. Wind 0—4 m.p.h.

Night time, therefore, is the most generally favourable time for employing gas ; and the hours of darkness favour the chance of effecting surprise.

#### (B) *Topographical Factors.*—

Practically all war gases used up to the present are heavier than air. Nevertheless, it is incorrect to suppose that as a result they will drain into depressions, hollows, unprotected



dug-outs, etc., except under exceptional circumstances. The reason for this is that the gas is mixed with air and the density of the resultant mixture, for ordinary concentrations, differs from that of the air by only a negligible amount. Near gas sources, which are discharging at a very high rate, *e.g.* in the immediate vicinity of a gas-filled mortar bomb, density has an effect, but in the case of a gas cloud drifting over a trench there is no density effect.

At night sometimes a wind blowing downhill may be brought about by a fall in the temperature of the air and a consequent increase in its density. Gas mixed with such air, will, of course, flow downhill.

War gases tend to persist in dug-outs, trenches, woods, ruins, etc., mainly because the sun and wind have less chance of dissipating them.

The type of terrain and temperature of the ground will affect persistence. For example, bare rock is particularly unfavourable to persistence because it does not absorb liquid to any extent. If the ground is cold, mustard gas will freeze on it. As the temperature increases vaporization will occur. These facts should be borne in mind when siting medical units.

**3. Concentrations.**—Any gas may exist in the air in a concentration so low as to be harmless. Mustard gas, however, if present in sufficient quantities to be detectable by the sense of smell, is dangerous; and even when not so detectable it may exist on the ground in quantities sufficient to produce a burn on contact.

The object for which gases are used, and the weapons by means of which they are dispersed, are described in Chapter II of Defence against Gas, 1927.

**4. Classification of Gases from the point of view of their Physiological Effects.**—Though poisonous gases and vapours may, under appropriate conditions of administration, have a widespread action throughout the body and affect a number of different organs simultaneously, they usually produce their most marked effect on some particular structure, especially when they are present in low concentrations. Their action, therefore, may in some sense be regarded as specific, and it is possible on this basis roughly to classify the gases used for offensive purposes during the Great War according to the most prominent lesions produced by them. Such a classification is by no means rigid, for each of the gases really partakes of the characters of two or more of the groups, but it has the merit of convenience. As the result of experience



it has become usual to adopt the following classification of warfare gases :—

(a) *Vesicants* (e.g. mustard gas and lewisite).—These are substances which, either in the liquid, solid or vapour state, will damage any part of the body with which they come in contact. The typical effects produced are acute conjunctivitis, inflammation of the mucous membranes of the respiratory passages, and inflammation of the skin, followed, perhaps, by the formation of blisters. They are essentially casualty producing agents. Deaths, when they occur, are rarely caused by the direct effects of the poison, but are the result of secondary bacterial infection of the damaged tissues.

(b) *Lung Irritants* (e.g. phosgene, chlorine, chloropicrin, etc.).—These are usually encountered in the gaseous state and exert their most marked effect upon the deeper respiratory passages, and in particular upon the alveoli of the lungs. This action results in the production of pulmonary œdema, which, if in sufficient amount, is liable to bring about a fatal termination. They also exert an irritant action on the upper air passages.

Some of these gases are also quite effective lachrymators in concentrations met with in the field. They are used primarily as lethal agents.

(c) *Sensory Irritants* (e.g. diphenylchlorarsine, diphenylcyanoarsine, etc.).—These are clouds of fine solid particles which have an effective action in low concentrations on the eye, nose and upper respiratory passages. They give rise to intense burning and aching pains in the eyes, nose, throat and chest, accompanied by nausea and extreme mental depression. The effects clear up rapidly, however, on withdrawal from the poisonous atmospheres. They are used primarily as harassing agents.

(d) *Lachrymators* (e.g. bromobenzyl cyanide, xylyl bromide, chloroacetophenone, etc.).—In extremely low concentrations these have an immediate and violent irritant action on the eyes, which causes a profuse flow of tears, much pain and smarting, and spasm of the eyelids, though the effects hardly outlast the exposure. In extremely high concentrations they may have an action on the lungs similar to that of the acute lung irritants.

(e) *Direct poisons of the nervous system or paralysants* (e.g. hydrocyanic acid gas).—In sufficient concentrations these act with great rapidity on the vital functions of the nervous system, death being a matter of a minute or two, or less.



(f) *Gases which interfere with the respiratory function of the blood* (e.g. carbon monoxide).—Such gases are not used primarily as chemical warfare agents, but cases of poisoning from such gases are not uncommon in war. These are true gases and owe their poisonous property to the fact that they cause an alteration in the hæmoglobin, or lead to the destruction of red blood corpuscles.

5. Broadly speaking the gases in groups (b) and (e) may be regarded as lethal agents, and those in groups (c) and (d) as irritants, capable of putting a man out of action immediately, though only temporarily; whilst those of group (a), though intensely poisonous, have, when used against troops who are well disciplined in defence against gas, a casualty producing power enormously in excess of their killing power.

---

## CHAPTER II

### VESICANTS

The only war gas of this group employed during the Great War was dichloro-diethyl sulphide, commonly known as "mustard gas" by the British, "yperite" by the French, and "yellow cross" by the Germans. Lewisite, which is dealt with later, in addition to its irritant action has pronounced vesicant action.

### MUSTARD GAS

**6. Chemical and Physical Properties.**—In the pure state mustard gas is a clear, almost colourless, heavy and somewhat oily fluid which has only a faint odour. It has a high boiling point,  $217^{\circ}\text{C}$ . ( $412^{\circ}\text{F}$ .), and becomes a white crystalline solid at  $14.4^{\circ}\text{C}$ . ( $58^{\circ}\text{F}$ .). It is heavier than water, the specific gravity being about 1.28 at  $15^{\circ}\text{C}$ . ( $59^{\circ}\text{F}$ .). It has a low vapour pressure; at  $10^{\circ}\text{C}$ . it is 0.05 mm. Hg. and at  $40^{\circ}\text{C}$ . ( $104^{\circ}\text{F}$ .) 0.45 mm., that is to say it vaporizes very slowly at ordinary temperatures, and so is termed a highly persistent substance.

In the crude form, as used in shell fillings, it is a heavy, dark-coloured, oily liquid. Compared with the pure compound the odour is more pronounced and it freezes at a considerably lower temperature, about  $7^{\circ}$  or  $8^{\circ}\text{C}$ . ( $44^{\circ}\text{F}$ . to  $45.4^{\circ}\text{F}$ .). Ground that is splashed by the liquid will, therefore, be a



source of danger for a time depending upon the conditions of the weather, and men may unknowingly carry the liquid on their boots or clothing into dug-outs and other shelters where it will vaporize in the warmer atmosphere.

**7. Outstanding Features.**—Mustard gas in its relation to chemical warfare has very definite outstanding features, whether regard is paid to its chemical and physical properties or to its poisonous qualities.

From the chemical and physical points of view the outstanding features are its stability, solubility in oils, its persistence and marked powers of penetration. As a poison the outstanding features are its odour, great toxicity, insidious action, delayed action, and delayed healing. These features will be considered in greater detail as follows :—

(a) *Stability.*—Mustard gas is, relatively speaking, a stable compound, and is not readily broken down into harmless products by substances ordinarily available, and so the question of decontamination or destruction presents a problem of considerable difficulty. Decontamination may be effected by any of the following methods, which must be chosen to suit the nature of the contaminated article: heat, that is driving it off in the form of a vapour; hydrolysis, in boiling water or steam; chlorination, the most convenient agent to use being bleaching powder; or by weathering, that is evaporation. (For detailed description of these methods, *see* Defence against Gas, 1927, Chapter V.)

(b) *Solubility.*—Mustard gas, both in the form of the vapour and the liquid, dissolves readily in many oils, such as fuel and lubricating oils, and in animal and vegetable oils and fats; it is soluble in many organic solvents, such as alcohol, petrol, mineral naphtha, etc.

Liquid mustard gas dissolves to a slight extent in water (under 1 per cent), but the amount thus dissolved is sufficient to render the solution dangerous to handle. If, however, such a solution is allowed to stand, the mustard gas is gradually hydrolysed into harmless products. This decomposition in water takes place more rapidly on warming, and at boiling point is complete in a few minutes.

(c) *Persistence and Powers of Penetration.*—Like an oil it penetrates most ordinary substances, but the special point to note is that it does so more readily. This marked power of penetration is a characteristic of the substance. Once it has penetrated, being a highly persistent substance, it will give off its toxic vapours over a period of time, depending upon the prevailing weather conditions.

(d) *Odour*.—The most important characteristic of mustard gas from the point of view of its detection in the field is its odour. At present the sense of smell is the only practical means of detection available, though droplets of the liquid and larger splashes may be visible to the eye. The sense of smell is the most powerful defensive aid against mustard gas, and it is, therefore, most important to train all those who may be brought into contact with it to recognize its odour.

Mustard gas has a faint but characteristic odour ; by some it is likened to mustard, and by others to horse-radish, onions or garlic. It is immaterial which particular substance the odour suggests to the individual, the practical point is that the smell must be memorized so that its presence can be detected in the field.

The vapour when breathed causes no immediate irritation. This fact constitutes one of its greatest dangers, as serious consequences, even resulting in death, may result from a long exposure to an atmosphere which has produced little or no discomfort at the time.

The odour may be detected on first entering a contaminated area, but, as with other substances, the sense of smell soon becomes dulled, and the odour may cease to be appreciated after a short time.

These are facts of importance, as the danger is only too likely to be underestimated in the absence of warning by a strong odour or immediate irritant effect. The sense of smell recovers, however, on breathing pure air or by wearing the respirator. An additional fact of importance is that the odour of mustard gas may be masked by the presence of more powerful smells.

(e) *Toxicity*.—Mustard gas, both in the form of the liquid and the vapour, is an extremely poisonous substance ; but although this is so, it is essentially a local poison, exerting its action only upon those tissues with which it comes in contact.

(f) *Insidious Action*.—Contact with the liquid or the vapour attracts no attention, droplets splashed on the skin do not betray their presence, as do more readily volatile substances, by a feeling of cold, nor is there smarting or other sensations, and so contamination may be unsuspected.

(g) *Delayed Action*.—There is always a period of delay before the onset of the signs and symptoms. This latent period depends chiefly upon two factors (1) the concentration of the gas, and (2) the length of time of exposure. The substance is cumulative in action, and hence exposures to



detectable concentrations over prolonged periods of time will eventually result in an injury.

(h) *Delayed Healing*.—One of the most characteristic features of an injury due to mustard gas, when it has penetrated the tissues deeply, is the marked delay in healing. The reason for this is not yet fully understood. The tissues are devitalized by the substance, and as a result are readily abraded by careless rubbing or pressure, and also form an excellent nidus in which organisms flourish. On the other hand, where the injury only amounts to a mild inflammation of the skin and ceases at that, or where blisters are only of a superficial character, the condition clears up in a matter of days, as distinct from deeper injuries which take a matter of weeks.

### 8. Factors influencing the Action on the Body.—

The respirator, if properly adjusted, affords complete protection to the eyes and the lungs. Protection of the skin is a much more difficult problem, since ordinary clothing readily absorbs the vapour and is penetrated rapidly by the liquid. Burns are caused not only by the direct action of the vapour on the skin while in the mustard atmosphere, but also by the mustard gas, which ordinary clothing readily absorbs. The clothing, therefore, if worn after leaving the mustard atmosphere, may be a source of danger. It has been shown experimentally that clothing, when greasy and damp with sweat, will absorb much more mustard gas than the same clothing when dry and clean. This is one explanation of the fact that in the late war the most severely burnt areas were the perineal region and the inner surfaces of the thighs, the axillæ, and the flexures of the elbows.

Some persons who have been burned by mustard gas show an increased and long-lasting sensitivity on further exposure to the poison.

If on exposure to the vapour of mustard gas the respirator has not been worn, or if it has been from time to time removed in the contaminated atmosphere, signs and symptoms affecting the eyes, the respiratory passages, and the skin begin after the lapse of anything from two to twenty-four hours, or even longer. The latent period before the onset of the signs and symptoms depends chiefly upon the length of time the individual has been exposed, and the concentration of the vapour. The higher the concentration of the vapour the shorter the exposure necessary to produce an injury, and the shorter the latent period. Conversely, the lower the concentration the longer the exposure necessary, and the longer the latent period.

The eyes are the most sensitive part of the body to the

action of the vapour, and consequently eye symptoms appear first. The respiratory passages are the next in the order of susceptibility; and lastly comes the skin.

The most important action of mustard gas on the body, from the point of view of the commander of a force, is its effects on the eyes. A man may be able to carry on his duties after a mustard gas injury to the skin has developed, but as soon as the eyes become affected, he is liable to become a helpless casualty. The majority of the mustard gas casualties which occurred in the Great War had eye injury, either alone, or together with inflammation of the skin and huskiness of the voice. Blisters were not so common. The most common mustard gas casualty seen at any of the dressing-stations in France was temporary blindness. This was due to the men not realizing that the gas can be dangerous even in the absence of a strong odour or an immediate irritant effect, and that frequent exposures to low concentrations of the gas will produce eye casualties. It cannot be too strongly emphasized, therefore, that where the odour of mustard gas can be detected there is danger. To obviate becoming a casualty from affections of the eyes, particular care must be taken to keep the respirator adjusted, in order to protect this relatively sensitive part of the body.

**9. Mode of Action.**—Mustard gas is a direct irritant to the skin and mucous membranes; there is no proof that its action is due in any way to absorption and conveyance by the blood stream to parts remote from the point of contact or exposure. Its high lipid solubility accounts for its rapid penetration into the tissue. Once it has penetrated within the cells it commences to exert its harmful effect, although, as has already been stated, the clinical signs do not make their appearance until the lapse of some hours.

**10. History of a typically severe and totally unprotected case.**—On exposure to the vapour, or even on contamination by the liquid, no signs or symptoms are experienced immediately. The only clue to contamination may be by detecting the faint though characteristic odour, or by actually seeing the liquid, or a stain. It must be repeated, there is no pain or other sensation in the early stages. After the lapse of two hours or longer, signs of injury may appear, without as yet any subjective phenomena, followed later by symptoms which subsequently develop with some rapidity.

The eyes are the first to show any signs of irritant action. They begin to smart and water, the ocular conjunctiva is injected, especially in the interpalpebral aperture.



The nose about this time commences to run with thin mucous, as from a severe cold in the head, and sneezing, which appears after the conjunctivitis and rhinitis have developed, is frequent.

Nausea, retching and vomiting associated with epigastric pain begin about the same time as the pain in the eyes, and recur at frequent intervals for several hours.

During the next few hours the conjunctivitis increases in intensity and the vessels are deeply injected. The throat feels dry and burning, the voice becomes hoarse, and a dry, harsh cough develops. Inflammation of the skin now shows itself in a dusky red erythema of the face and neck, which look as though they had been scorched, while the areas which are moist and greasy, as the axillæ, inner aspect of the thighs, genitals and other sheltered parts of the body are similarly affected. Should any part of the clothing have been actually splashed by the liquid, or have been contaminated with impregnated earth, etc., the underlying skin will be profoundly inflamed. During the stage of inflammation, the patient may suffer from the most intense itching, particularly if he gets hot, which interferes with rest and sleep and is most wearing. The injury to the skin may amount to a mild degree of inflammation and cease at that, but where the effect of the poison is more serious small vesicles may appear which quickly coalesce to form large blisters in the same area.

At the end of twenty-four hours a typical appearance is presented. The main distress is caused by the pain in the eyes, which may be very great. The patient is unable to see on account of the inflammation and œdema of the conjunctiva of the eyelids. Tears ooze between bulging, œdematous eyelids over his reddened and slightly blistered face, while there is a constant nasal discharge and an occasional harsh, hoarse cough. The respiration is fairly normal both in rate and depth. There may be frontal headache associated with the pain in the eyes and photophobia, and blepharospasm is always a marked symptom. Death practically never occurs during the first twenty-four hours.

During the second day the condition is aggravated by the development of the vesicles into large blisters over the deeply erythematous areas, where the inflammation is obviously severe, while the scrotum and penis become œdematous and painful. Acute bronchitis now sets in, with abundant expectoration of muco-pus, in which may be found actual large sloughs from the inflamed tracheal lining. An early indication of involvement of the respiratory system is an increase in the temperature, and in the pulse and respiration rates. Secondary infections of the necrosed mucous membrane in

the respiratory tract soon lead to the development of a broncho-pneumonia, with slight cyanosis and cardiac dilatation. Death may occur at any date from the second or third day to the third or fourth week in the more lingering cases, the highest death rate occurring at the end of the third or fourth day after exposure. Rarely, abscess of the lung, bronchiectasis and even gangrene of the lung supervene. The death-rate with well-disciplined and well-protected troops is low, approximating to two per cent of cases, since the respirator will protect the respiratory tract; but with ill-protected troops it may be very high.

### 11. Main Features of Casualties.—

Insidious mode of onset, with delay of obvious effects for at least two or three hours and often longer.

Conjunctivitis varying from mere injection of the conjunctiva to, in rare cases, inflammatory changes of an extreme degree.

Huskinness of the voice, appearing within a few hours of the exposure and almost simultaneously with the conjunctivitis.

Nausea, retching and vomiting associated with epigastric pain coming on as early as the conjunctivitis, and being persistent and intractable.

Erythema of the exposed surfaces, and more especially of the moist protected skin areas, followed later, in severe cases, by blistering or excoriation due to devitalization of the tissue.

Marked delay in the healing of severe blisters, which eventually do heal, leaving a temporarily pigmented scar.

Inflammation of the trachea and bronchi, with perhaps necrosis of the mucous membrane leading to the development of secondary bronchitis or broncho-pneumonia.

**12. Morbid Anatomy.**—The most important pathological changes are those in the respiratory tract. There is intense inflammation of the mucous membrane from the anterior nares to the finest ramifications of the bronchioles, which show in varying degree the caustic effect of this acute irritant. The destruction of the membrane may have proceeded to such an extent that the whole area of the trachea and the larger bronchi is covered by a loosely adherent slough of a yellow colour several millimetres thick. Occasionally this slough can be separated as a whole and removed, giving the appearance of a cast of the bronchial tree. On removing this slough a raw granulating surface is exposed, which may, in late cases, be pitted with minute ulcers.

The interior of the nose and naso-pharynx may exhibit a similar pathological change.

When the chest is opened small petechial hæmorrhages



may be observed on the pleural surface, but it is not common to find much pleural exudation. In cases where death has taken place early, the lungs, on section, will be found to contain areas of emphysema alternating with small patches of collapse, and small hæmorrhages and areas of inflammation will also be visible to the naked eye. The lung, however, on section is dry, and in this respect forms a marked contrast to the post-mortem appearances following lung irritant poisoning, in which the lungs are saturated with œdema fluid, which pours out on section.

In cases which have survived to a later period, in addition to the appearances described above, which are to be regarded as the result of inflammatory reaction, the signs of a secondary septic infection appear, for as a result of the intense irritation of the bronchial tubes, pus is secreted, which in gravitating downwards carries with it micro-organisms and sets up new foci of broncho-pneumonia, whilst intensifying those caused by the direct action of the mustard on the terminal bronchioles. In late stages, indeed, the whole of the lower lobes may be practically solid from the confluence of numerous patches of this broncho-pneumonia. Owing to their septic origin, many of the patches tend to break down and form small abscess cavities. On section of such a lung, pus will be seen to exude from the fine bronchial tubes. As a result of the patches of broncho-pneumonia the intervening areas of lung which are not consolidated are frequently œdematous, but this œdema is a secondary and local effect in contrast with the primary and general condition induced by the lung irritants.

The skin exhibits all stages of burn from the primary erythema up to the final stage of a deep burn with necrosis and sloughing of tissues. The latter condition, of course, is only seen where life has been maintained for a considerable period, and ordinarily in the fatal cases the skin lesions will not have proceeded beyond the stage of vesication and stripping of the epidermal layers.

A mustard burn differs from a burn caused by heat in the following respects :—

- slow and progressive development of the lesion ;
- a more intense inflammatory reaction ;
- marked delay in the process of repair ; and
- greater tendency to septic infection.

In the case of the mustard gas burn there is no immediate naked eye evidence, but histological evidence of the action of mustard gas on the skin may be apparent within a few minutes of its application. This is seen as shrinking and pyknosis of the nuclei of the cells, followed by engorgement of the superficial capillaries, œdema of the connective tissues,

and diapedesis of leucocytes. These changes in the tissues progress until the resulting inflammation becomes apparent to the naked eye. This inflammation is of greater intensity than in the case of a thermal burn.

During the stage of erythema, resulting from mustard gas, the affected area is always moister than it is when caused by heat, and when the erythema has given place to the formation of a vesicle there is an intense exudation of fluid, which may according to the degree of penetration of the mustard gas continue for several days.

Vesication, as the result of exposure to concentrations of mustard gas ordinarily met with in the field, usually begins with the formation of minute vesicles, which make their appearance after the lapse of twelve hours or longer, and these run together to form a large vesicle on the second day.

In more severe burns there is a progressive necrosis of the dermis which reaches its maximum in about five to ten days. The depth of the burn will depend largely on the penetration of mustard gas, which may be assisted by the hair follicles and the ducts of the sebaceous and sweat glands. This may explain why those parts of the body which are naturally moist and greasy, having a more abundant gland supply, are usually more severely affected.

A severe mustard gas burn never has the charred, coagulated appearance of a thermal burn. In a thermal burn there is an immediate destruction of tissue, and following a short period of inflammatory reaction there ensues a process of repair which consists essentially in the demarcation and delimitation of the necrosed tissues by a zone of leucocytes.

This essential process of repair is much delayed in the case of a mustard gas burn, and even after necrosis has ceased there is evidence in the microscopical appearance of the tissues, more particularly of the inflammatory exudate and of the young connective tissue, which suggests that a subacute inflammatory focus is still continuing.

The tendency to hæmorrhage from the newly formed capillaries also supports this view.

Furthermore, the granulation tissue is not so healthy, the fibres appear to be separated by fluid, the tissue is poorly supplied with capillaries, and regeneration of the surface epithelium is much delayed. Healing of the burn results in the formation of a paper-like cicatrix which is usually pigmented.

There is, therefore, a histological explanation for the fact that mustard gas lesions form an excellent nidus for the growth of septic organisms; septic invasion and sloughing is a common feature, and such areas are readily abraded by pressure or rubbing.



The eyes share in the general inflammation of the skin and exhibit all stages of an acute conjunctivitis from the early chemosis up to ulcerative keratitis, which, on becoming secondarily infected, may cause hypopyon, perforation or panophthalmitis. These complications are, however, rare.

The abdominal organs show very few lesions. The mucous membrane of the stomach may be injected and contain a few small hæmorrhages. The intestinal tract, as a rule, escapes entirely, although occasionally the upper part of the small intestine shares in the inflammatory changes seen in the stomach.

There are no characteristic changes to be seen in the kidneys beyond the congestive swelling and, occasionally, on stripping the capsule, subcortical hæmorrhages.

The blood shows no special changes. Some observations suggested a decrease in the coagulation time. There is an early leucocytosis, especially of the polymorphs, and a leucopenia when the case is drifting to death. The figures for the blood-count are very similar to those for ordinary broncho-pneumonic infections of the lungs.

Punctate hæmorrhages have occasionally been seen in the white matter of the brain, but this condition is by no means so common as in phosgene poisoning, where profound cyanosis affords evidence of a degree of oxygen starvation far greater than that seen in mustard gas poisoning.

13. *The main pathological lesions which cause death are, therefore, as follows :—*

An acute inflammation of the air passages from the larynx downwards, followed by entire desquamation of the mucous lining, with a remarkable formation of false membrane. This membrane, in addition to the actual obstructive effect it produces, affords a suitable pabulum for organisms. An acute purulent capillary bronchitis is superadded, leading to collapse of lung tissue and then to broncho-pneumonia.

Further, the effect of burns leading to a general septic infection will be found in the late cases, and occasionally an inflammation of the mucous membrane of the large intestine.

The microscope bears out the naked eye observations, the lesions in the air passages being essentially those caused by a necrosis of mucous membrane followed by a secondary broncho-pneumonia.

#### 14. Detailed effects on Individual Organs and Tissues.

—*The Eyes.*—The individual feels no discomfort at the time of exposure. The part of the conjunctiva covered by the lids is never severely affected. Neither necrosis nor ulceration of the conjunctiva occurs unless actually splashed by liquid

mustard gas. The symptoms in all cases come on slowly, on an average from six to ten hours after exposure to the gas. The onset may be delayed for a very long period, and then is probably due rather to the cumulative effect of repeated small doses than to one single exposure. Men, for instance, who have been living in contaminated dug-outs for a long time and who have occasionally been forced to remove their masks will, on each such exposure, receive a minute dose, which ultimately will be sufficient to cause effects.

In the slight cases there is severe stinging in the eye and lachrymation, the lids cannot be raised, the ocular conjunctiva becomes injected, especially in the outer palpebral aperture, but the cornea is not affected. In the more severe cases the pain is acute, with blepharospasm and profuse lachrymation; also there is much greater reaction, the skin of the lids becomes puffy, the conjunctiva in the interpalpebral aperture swollen and red and may project between the lids, but the cornea is not visibly affected. In the severest form the symptoms are very serious. There is intense and burning pain in and about the eyes associated with marked blepharospasm, and the eyes cannot be opened. There is profuse lachrymation between the closed œdematous lids, the skin of which may be blistered. The conjunctiva in the interpalpebral aperture, although only slightly swollen, has an opaque, yellowish-white colour due to coagulative exudate in its substance. This exudate presses on and obstructs the capillary circulation, thus presenting a dead white band on either side of the cornea. At the same time the ocular conjunctiva under the lids, which is much less affected, becomes greatly swollen, so that two chemotic folds may be seen protruding between the lids, and to the inexperienced observer it may be thought to be more severely affected. The cornea in the exposed area, in the early stage, is grey and hazy; above this area its surface usually remains bright. Where grey, its surface is dull, stippled and roughened, due in the first instance to œdema of the epithelium and later to actual exfoliation of cells. The sensation of the cornea to tactile stimuli is often lowered and may remain so for weeks.

The condition of the cornea calls for the most careful and regular examination throughout, and it may be necessary to use a lid retractor where there is much swelling or blepharospasm.

Ulceration in gassed eyes may result from exfoliation of the damaged epithelium, or from an abrasion. Exfoliation and abrasion are especially liable to progress to definite ulceration in the presence of secondary infective conjunctivitis, with mucopurulent discharge. An ulcer under these circumstances



readily becomes infected and infiltrated, leading to permanent opacities and impairment of vision.

In severe cases, therefore, impairment of vision may result from :—

- (a) Diffuse nebulæ of a temporary nature which may occur without definite ulceration.
- (b) Ulceration, leading to opacities.
- (c) Ulceration, which on becoming secondarily infected may cause hypopyon, perforation and panophthalmitis.

Where actual splashing with liquid occurs, an infiltrated ulcer of the cornea may appear early and may be one-sided.

In the mild cases, which are by far the most common type seen, resolution is rapid, and if the mental and physical tone are not lowered, under appropriate treatment the individual is usually fit to return to duty within a fortnight. In the more severe cases, especially where the cornea is affected, the period of convalescence may be prolonged to several weeks. The chemotic folds above and below the cornea subside in a few days and the ocular conjunctiva covered by the lids gradually pales and becomes white. The solid œdema in the interpalpebral aperture often takes a week or ten days to absorb. Vascularization usually spreads in from the upper and lower sides of the triangular patch until the whole white area disappears and the conjunctiva in the interpalpebral area becomes red. This stage is almost identical with the early stage of the mild burn.

The two symptoms liable to persist are lachrymation and photophobia. There is a tendency for a neurasthenic condition to supervene, and unless this is firmly treated convalescence may be unnecessarily prolonged.

**15. Respiratory Tract.**—Inflammation of the mucous membrane of the nose is accompanied by profuse watery secretion, which later may be purulent and associated with the separation of large sloughs. Ulceration and epistaxis are rare.

Inflammation and erosion of the posterior pharyngeal wall may be sufficient to interfere with swallowing. The larynx is sometimes œdematous, but never so seriously affected as to necessitate tracheotomy. Later, there is a mild laryngitis or even ulceration. Prolonged exposure to a very low concentration of the vapour may often cause laryngitis, with loss of voice, to develop slowly even when there is no conjunctivitis at all.

The main pathological features in the trachea, bronchi and lungs have already been described. Death may occur solely

from the action of the poison on the respiratory tract ; and in severe cases it may ensue in thirty-six hours from the necrosis, or even be asphyxial by blockage of the lower air passages with loosened membrane, before secondary infections have had time to develop.

For the first twenty-four hours there may be only hoarseness of voice, substernal pain and a slight cough, but soon the rising temperature and quickening pulse-rate herald the onset of infection leading to bronchitis and fatal broncho-pneumonia. In the more protracted cases there is profuse thin purulent expectoration, and at autopsy the septic pneumonia is found to be confined to the lower lobes.

16. *The Skin*.—Skin lesions may be caused either by direct contact with liquid mustard gas, as in splashes from shell burst, by handling leaky shell, or from contact with infected earth, etc., or by the vapour which diffuses through clothing. The distribution of the injury will vary accordingly. As noted above, moist and greasy parts of the body are the most sensitive to the irritant action of the vapour.

The successive effects are :—

(a) A diffuse erythema which may appear in from two to forty-eight hours, and tends to spread rapidly, or may be delayed, either locally or generally, for several days.

(b) Very superficial blistering over erythematous areas, usually at first as small vesicles which ultimately coalesce into large blebs. These develop as painless collections of yellow or reddish serum just beneath the epithelium. If the latter is removed a raw weeping surface is left, and such burns may then become the seat of secondary skin infections and lead to a very painful eczematization. This is particularly likely to occur in blisters of the scrotum and penis. The blisters may appear on the second day, but there may be an outcrop even in the week following exposure and long after the patient has been carefully washed and his clothing changed.

(c) In the severest cases, the bright red colour of the inflammation rapidly gives place to a bluish, violet or brown colouration, while the swelling of the epidermis, cutis, and even subcutaneous tissue distinctly increases at the same time. These changes occur in the centre of the area of erythema within eight to twenty-four hours after exposure. Blisters appear on the dark background. Their removal reveals a surface which is at first deep red in colour and slightly hæmorrhagic, but soon becomes moist with viscous purulent exudation. These lesions progress slowly, the ulceration may spread beyond the limits of the original blister, and six to



eight weeks, or even longer, may elapse before they are completely healed.

(d) Staining of the skin with a dark brown or brownish-purple tint usually occurs in areas that had previously been erythematous. The staining is of no consequence, and it vanishes in three or four weeks as the cuticle desquamates.

The majority of skin lesions from vapour contamination heal fairly rapidly, and unless they become septic there should be no residual trouble at the end of a month. In severe burns, healing may be considerably delayed beyond this period. All the lesions are very liable to infection by pyogenic organisms.

17. *Alimentary Tract.*—The early vomiting due to the swallowing of saliva or nasal secretion, which has been infected with mustard gas, rarely persists for more than a day, and the epigastric pain vanishes shortly afterwards. Hæmatemesis is exceedingly rare. There are no lasting after-effects in the stomach, and the bowels are not usually affected.

18. *Urinary Organs.*—Albuminuria has been described in the first twenty-four hours in cases of early fatality, but it is not found at a later date. Very rarely an acute hæmorrhagic nephritis has been observed. Pain on micturition and even retention of urine may result from œdema and blistering of the penis.

19. *Circulatory System.*—The blood is unaltered ; the heart is unaffected at first, except by the changes associated with pulmonary infection. During early convalescence some of the cases complain of headache, giddiness after exercise, and of shortness of breath associated with precordial pain and tachycardia. The symptoms are chiefly due to a nervous debility, and they yield quickly to tonics and graduated exercises under firm discipline.

**Treatment.**—This can be discussed under two heads :—

A. PREVENTIVE.

B. CURATIVE.

20. *A. Preventive.*—It cannot be too strongly emphasized that in the prevention of mustard gas injuries, the first essential is efficient gas reconnaissance which presupposes the detection of the gas by its odour at the earliest possible moment. It must be repeated that the respirator, if properly adjusted, affords complete protection to the eyes and to the respiratory system. But no matter how good the gas discipline may be, or how efficient the respirator, it must be remembered always that if the individual is exposed to the vapour of mustard gas he is liable to become a casualty from

skin lesions. Frequent exposures to mustard gas, even though in relatively low concentrations, may result in the production of an injury. In this connection attention is directed to the fact that ordinary clothing absorbs the vapour of mustard gas, and this may be an additional source of danger. Airing of the clothing in the open, however, readily removes any vapour. To avoid unnecessary casualties it is essential to distribute men, so far as the tactical situation permits, so that the minimum number are exposed to the action of the vapour.

Protective clothing has only a limited value in the field on account of the difficulty of obtaining gas-proof garments in which men can do heavy work.

There are, however, certain articles commonly available in the field which give a measure of protection; such, for example, are the cape ground-sheet and sand bags. The cape ground-sheet is not primarily designed or issued for the purpose of protection against contact with contaminated ground, but it may be used for this purpose. It will act as a protection against such contact for about one hour. When a ground-sheet is used in such a manner it requires subsequent decontamination.

The use of the cape ground-sheet does not eliminate the vapour danger from the surrounding area.

(For uses and methods of wearing protective clothing *see* Defence against Gas.)

Boots of the ordinary service pattern in good condition will protect adequately from contact with mustard gas, especially if the uppers have been treated with grease or dubbin.

Mustard gas, as soon as it comes in contact with the body, begins to exert its harmful effect, although, as has been stated, there is always a period of delay before the signs and symptoms manifest themselves. Time, therefore, is the factor of paramount importance in the preventive treatment, and no time should ever be lost in resorting to preventive measures. The preventive treatment consists essentially in the complete removal of the clothes of the contaminated person, and in cleansing the skin. If, however, the individual has been subjected to only a low concentration of the vapour, or to a high concentration for a comparatively short time it may not be necessary to remove the clothing; similarly, it may not be necessary to cleanse the skin; each case will require to be considered on its own merits. Cleansing of the skin may be carried out either by a process of mechanical removal of the mustard gas, or by chemical destruction by means of bleach. Mechanical removal is best carried out by scrubbing, preferably



in a bath with soap and water, using frequent changes of water, and taking not less than five minutes over the operation of scrubbing. The facilities for doing this are seldom available in the field, and therefore, as an alternative method to bathing the following preventive treatment may be employed with as good results: immediately rub well into the affected area of skin bleach either in the form of a cream or an ointment composed of two parts of bleach to one of vaseline. The bleach should be left undisturbed for two or three minutes and then wiped off as thoroughly as possible. Used in this manner it has little irritant action on the skin, but care should be exercised in preventing it getting into the eyes, or allowing it to remain indefinitely on sensitive areas, as it can produce itching and redness. If rubbed on the skin immediately after exposure to mustard gas vapour or liquid, it diminishes the subsequent effects. It is not desirable to apply this ointment as a protective measure before exposure, since the protective effect diminishes rather rapidly, and bleach would give rise to irritation if left on the skin for any length of time. As time is the factor of paramount importance in the cleansing of the skin after mustard gas contamination, it is necessary for personnel exposed to such contamination to have the remedies described above immediately available and to apply them at once.

Should it not be possible to deal with the contaminated person until some time has elapsed after contamination, thorough washing should still be carried out at the first available opportunity to lessen the degree of the burn. Bleach should be applied with caution once an erythema has developed as it may aggravate the condition.

If the eyes have been contaminated they should be bathed thoroughly with clean water, or normal saline, and a drop of liquid paraffin or castor oil should then be inserted to prevent the eyelids sticking.

21. *B. Curative.*—The essential is a two-fold one:—(a) to relieve immediate symptoms; (b) to prevent septic after-effects. Mustard gas kills not by its direct action, but because the inflammation it causes opens the door to a secondary invasion by the organisms which are common to the area affected. The question of treatment will, therefore, be best dealt with by taking in succession the various parts affected.

(a) *The Eyes.*—It is very necessary to reassure the individual at the earliest possible moment that the sight is not lost.

*Mild Cases.*—Bathe the eyes frequently with bland lukewarm lotion, such as boric acid, normal saline or 2 per cent sodium bicarbonate. Drop liquid paraffin into the eyes three times a day. If liquid paraffin is not available, castor oil can be used, but it is somewhat irritating.

Severe Cases.—Where there is marked blepharospasm and continued pain, insert sterilized 1 per cent atropine ointment between the lids at the earliest opportunity. Where the cornea is affected, in addition to frequent bathing, the regular use of atropine is very important and should be repeated sufficiently to keep the pupil well dilated, and continued until the cornea becomes quite smooth. While the pupils are dilated with atropine a shade should be worn, but the eyes must never be bandaged.

Cocaine should not be used, for its anæsthetic properties are transient, while its use leads to exfoliation of the corneal epithelium.

In such cases the nutrition of the cornea is always lowered, and secondary infection is liable to occur. It is important to remember, therefore, that such cases should be handled with the utmost care. Lotions, etc., which might have an irritant effect should be used with caution, and pressure, such as might be caused by a bandage, should never be applied.

If there is any catarrhal or muco-purulent discharge, a mild antiseptic should be instilled. A 2 per cent solution of argyrol or protargol instilled twice a day is very suitable. This is particularly important when the cornea is grey or roughened, to prevent an infiltrated ulcer. When ulceration occurs the same measures should be adopted, but stricter care must be exercised to keep the conjunctival sac clean by gentle and frequent bathing combined with very hot applications over the closed lids four times a day. If the ulcer becomes infiltrated it should be cauterized by the light application of pure carbolic acid put on with a nearly dry brush slightly moistened with the liquid. It must be borne in mind that in the already impaired conditions of the cornea too free application of another caustic might cause extensive necrosis and perforation. If hypopyon supervenes and does not clear up with hot bathing, atropine and cleansing of the conjunctival sac, Sæmisch section is indicated.

As soon as the cornea again becomes smooth, or, in milder cases, as soon as the primary irritation and swelling of the conjunctiva has subsided, atropine may be dispensed with, and astringent drops instilled three times a day, *e.g.* boric acid, gr. 10, zinc sulphate, gr. 1 to 2, adrenaline, drachms  $\frac{1}{2}$  to 1, and water to 1 oz.

When the pupil regains its normal size, the shade or dark glasses should be removed irrespective of the wishes of the patient. Such protections in the later stages of convalescence only accentuate and prolong the neurasthenic symptoms. The normal balance should be restored by fresh air and stimulating treatment, mental and physical. Experience during the



Great War showed that at least 75 per cent of the cases were mild and became fit for duty within an average of two weeks, 15 per cent had considerable conjunctival inflammation but no apparent changes in the cornea, and were fit for duty in four to six weeks, while 10 per cent had severe conjunctivitis with definite corneal changes, and were not fit for duty under two to four months.

(b) *Respiratory Tract.*

Rhinitis.—A warm alkaline douche should be poured, not insufflated, through the nose three times a day.

Laryngitis.—Steam should be inhaled from a pint of boiling water with which is mixed a teaspoonful of a mixture of tinct. benzoini co. 1 ounce, menthol 10 grains. The laryngitis should be completely cured in a fortnight. It is apt to be followed by a functional aphonia that requires the ordinary strict methods of treatment.

Tracheitis.—At first this may be eased by breathing through a Burney-Yeo's mask moistened with drops of some antiseptic mixture, *e.g.* menthol, grs. 20, tincture of iodine, minims 30, oil of eucalyptus, minims 20, creosote, drachms 1, chloretone, gr. 1, alcohol to 1 oz.

Broncho-pneumonia.—Venesection or oxygen may be useful in the cyanosis of secondary broncho-pneumonia, but such treatment is never needed in the early stage of poisoning by mustard gas. Treatment should be on the usual lines. The bronchitis clears up in a month or less in the non-fatal cases, and the use of expectorant mixtures should not be continued after the sputum has ceased to be purulent.

(c) *Alimentary Tract.*

Epigastric pain.—Warm draughts of a weak solution of sodium bicarbonate may be given to relieve pain. There is no need to induce vomiting.

(d) *The Skin.*

From the description given above (*see* page 20), of the histological appearances of a mustard gas burn, it will be appreciated that treatment presents difficulties that are not found in the case of a thermal or chemical burn.

Although many therapeutic agents have been given extended trials it has not yet been found possible to neutralize the toxic effect, or to limit the persistent and progressive action, of mustard gas once it has penetrated into the tissues.

Once the skin has been definitely penetrated a lesion will follow, and hence, treatment is directed to minimizing the

extent of the injury, to relieving the immediate symptoms, and preventing septic after-effects.

In order to minimize the extent of the burn it is always important to cleanse the skin as thoroughly as its damaged condition permits, to clip short all hair on the affected areas, and if a blister is present, to remove the raised epithelium, adopting strictly aseptic and antiseptic precautions.

Stage of Erythema.—The first thing which may call for attention is to allay the irritation so frequently present, especially in areas of more or less generalized erythema.

The following are useful for this purpose:—

- (a) Evaporating lotions.
- (b) Anæsthesin, which is described later, has been found useful in allaying the irritation when dusted lightly on the reddened skin.
- (c) Potassium or sodium permanganate lotion, 5 per cent.

Dusting powders are disappointing, and so also are ointments, unless they have definite analgesic properties. Erythemas of a moderate degree clear up in the matter of a few days, but where the erythema borders on vesication, the inflamed skin becomes very fragile, and the surface area is readily loosened by pressure or careless rubbing. Care, therefore, must be taken to protect such areas.

Stage of Vesication.—Blisters at their commencement may be in the form of tiny bullæ, which later may coalesce into areas many inches across. The blisters are usually quite superficial and almost painless in their development, but the raw surface which is left after the blister has burst becomes sensitive to all forms of irritation. To allay this pain, which may be most acute, an anæsthetic may be desirable. The choice of anæsthetic, and indeed, the choice of any drug for application to the skin where extensive areas are involved, requires consideration, owing to the rapidity with which absorption may take place. For small circumscribed blisters, the substance anæsthesin, which is the ethyl ester of p-amido-benzoic acid and which belongs to the orthoforms, is an excellent and non-toxic anæsthetic. It is best applied in the form of a dusting-powder from a dredger.

The nature of the treatment to be employed depends upon the extent of the area blistered and the depth to which the skin has been affected.

For small circumscribed blisters of a superficial character, potassium and sodium permanganate solutions 5 per cent, or silver nitrate  $2\frac{1}{2}$  per cent, or tannic acid solutions  $2\frac{1}{2}$  per cent, may be used. These substances, which act by forming



a coagulum on the raw surface, protect from secondary infections, and prevent absorption of toxic products into the general circulation. The number of applications to be made depends on whether the coagulated surface remains intact and dry. On the appearance of oozing the application must be repeated. If sepsis supervenes fomentations and moist dressings must be applied.

No opportunities have arisen since the Great War for trying out the effects of the tannic acid method of treating extensive blisters which have involved the deeper layers of skin, and where the exudation has been of a pronounced character continuing for several days. Experience in the Great War showed that aqueous dressings, such as picric acid, boric, normal saline, or Eusol irrigation were of benefit; magnesium sulphate has also been shown to be a useful dressing. There are certain disadvantages to some of these remedies; for example, Eusol irrigation is too painful as a rule, so also is magnesium sulphate without the use of a preliminary local anæsthetic. Any treatment adopted must have a definite antiseptic value as septic infection and mild sloughing are so commonly associated with blisters due to mustard gas. Care must be taken in the selection of drugs for treating extensive raw areas, owing to the rapidity with which absorption may take place; for example, picric acid cannot be used in extensive burns, or for any length of time for smaller burns, on account of the possibility of toxic absorption.

As a dressing lint is preferable to gauze as it is less painful to remove. Oiled silks keep in the discharges, increase irritation and make the wound sodden. Where the injured areas are extensive and painful, and where there is much sepsis and sloughing, a continuous bath, soaking the whole affected part, if possible, will be found a treatment both soothing and efficacious. A Eusol dressing, kept damp by a constant drip or a cotton wick feed, is also a useful method of treating the septic areas, although it may be somewhat painful. Continuous irrigation with normal saline is more comforting.

For the dry, somewhat harsh and scaly condition of the skin, which is a fairly frequent sequela of mustard burns, especially of the generalized erythema short of actual vesication Lassar's paste is a useful application (Lassar's paste: zinc oxide (finely sifted), 3 oz., salicylic acid in powder, 140 gr., soft paraffin, white, 8 oz.), or a mild ointment such as the following: boric acid, gr. 15, powdered lead acetate, gr. 5, zinc oxide, gr. 20, paraffin to the oz.

It will be seen that the treatment indicated above follows

the line of that adopted for thermal burns, but is modified by the following factors :—

1. The oozing of serum from the surface for several days.
2. Greater liability to sepsis owing to devitalization of tissue.
3. Marked delay in healing.

For these reasons the results are often disappointing.

**22. General Treatment.**—In the first few days after exposure a light diet is desirable in all cases. Fluids, especially milk, may be given freely, and patients should be encouraged to drink water. No case should be moved if there is commencing fever, as this indicates the probability of broncho-pneumonia. As convalescence proceeds, a full diet is required, and this should be as varied as possible. The severe cases of pneumonia will naturally be kept on a fever diet, whilst patients with body burns require a generous diet and stimulants. The management of the convalescent periods provides the great test of the medical officer's ability, because he is required not only to treat the disease, but to restore morale, to cut short hospitalization, and to lift men out of the slough of self-analysis which so often follows gassing. As soon as a man is convalescent and free from the danger of septic complications, he should be discharged from hospital to a convalescent centre, where a well-ordered routine of exercise, employment, amusement and rest will quickly restore him to a state of physical and mental fitness.

**23. Functional After-Effects.**—This subject is of great importance from the point of view of wastage and duration of invalidity.

Functional disorders fall, in the main, into two classes. In the first, exposure to gas, often to a minimal and barely a toxic concentration, may yet prove the final factor in upsetting a nervous system already breaking down as the result of physical or mental strain. In such circumstances it will tend to produce an "anxiety state," similar in all respects to the neurosis so common in the war.

The second class is a far more important one, because in these cases a local but real organic lesion from mustard gas causes certain irritant reflexes, such as coughing or photophobia, and these sensory reflexes are perpetuated by introspection, almost in a form of conversion hysteria, long after their organic cause has been cured. Lack of appreciation of this possibility will cause much delay in returning men to duty.

Functional photophobia and aphonia are responsible for the great majority of cases. This is not surprising when it is



realized that the initial trauma affects a highly organized special sense, and that fear of blindness or dumbness resulting from the injury may very well act to perpetuate the symptoms. Ill-advised and unnecessary treatment is also a probable factor in many cases. The acute conjunctivitis with blepharospasm and photophobia that characterize the early stages of the eye lesion are naturally treated by protecting the eye from light. Eye shields are almost universally applied at casualty clearing stations, but they must not be retained in use after the necessity passes and actual manifestations of the eye lesions totally disappear. There can be no doubt that the suggestive influence of wearing a shade will perpetuate the functional manifestation.

Persistent aphonia, accompanied often by a useless, harsh cough, is another striking manifestation of auto-suggestion arising from the initial laryngeal irritation. The characteristic cough is either dry or accompanied by watery sputum, mainly of salivary origin; it is usually much worse at night, and is of a ringing, harsh quality.

The functional aphonia yields very rapidly to treatment by suggestion and breathing exercises, when the medical officer realizes the nature of the condition and gives the patient confidence in his early recovery.

Of all after-effects, functional or organic, those which seem to affect the heart present the greatest difficulty in assessment. Shortness of breath and tachycardia from exercise, *i.e.* "the effort-syndrome," or D.A.H. arise from so many diverse causes that gassing naturally was regarded as one of them.

It is clear, as the result of experience in the Great War, that under competent medical treatment the incidence of D.A.H. in mustard gas casualties should be very low when serious complications, such as broncho-pneumonia, are absent, and that such an after result is not to be feared with mustard gas to anything like the same extent as it is with phosgene poisoning.

The treatment of the features of D.A.H. in mustard gas casualties is by graduated and carefully controlled exercises.

### Lewisite

24. Lewisite is a substance which was isolated by the American Chemical Warfare Service. It has not yet been used in war. Its chemical name is chlorovinyl dichloroarsine, and it is thus analogous to the arsenical sensory irritants which are described in Chapter IV.

As, however, in addition to sensory irritation it produces burns like mustard gas, it is classified among the vesicant substances.

25. Lewisite is a liquid at ordinary temperature. It freezes at  $-13^{\circ}$  C. and boils at  $190^{\circ}$  C. Its vapour pressure, 0.396 mm. of mercury at  $20^{\circ}$  C., is comparatively low, so that it may be regarded as a persistent substance. It is soluble in oils, benzene, and in ordinary organic solvents. In contact with water it hydrolyses rapidly; this action is hastened by elevated temperature and the presence of alkalies. It has an odour which resembles that of geraniums.

Both in its liquid and vapour form it produces lesions similar to those due to mustard gas; but it is not so powerful in its effects. Thus, in the case of skin blisters, the injury does not extend so deeply and heals more readily under appropriate treatment.

The delay in appearance of the injury is not so long as in mustard gas, and erythema may appear within twenty minutes of the application of the liquid to the skin. Individuals appear to vary considerably in their sensitivity to this substance.

A lewisite burn, if extensive, may give rise to systemic symptoms of arsenical poisoning. The presence of arsenic can be demonstrated in the blister fluid.

26. *Preventive Treatment.*—The service respirator is a complete protection for the eyes and respiratory passages. Should injuries occur through failure to wear it, they should be treated on the lines laid down for mustard gas poisoning.

Success in the prevention of skin burns depends upon the promptness with which treatment can be applied. Thorough washing for five minutes with soap and water up to twenty-five minutes after exposure to the liquid is effective.

27. In the treatment of a lewisite burn it is essential that the blister be removed as soon as possible, the contents evacuated and the raw surface thoroughly irrigated, in order to minimize the danger of arsenical poisoning. The burns heal rapidly under a wet dressing of normal saline or a mild antiseptic, unless bacterial infection occurs. In the latter case they follow the ordinary course of septic sores.

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## CHAPTER III

### LUNG IRRITANTS

28. The acute lung irritant gases used during the war include phosgene, chlorine, chloromethyl chloroformate, trichloromethyl chloroformate, chloropicrin and phenyl-carbylamine-chloride. After the initial groups of cloud gas attacks, in which chlorine only was used, it was the almost invariable occurrence to find two or more gases of this group figuring in any cloud gas attack or gas shell bombardment.

29. All lung irritant gases cause essentially the same type of pathological effect, this being most pronounced on the alveoli of the lungs and the smaller bronchial tubes, and the great danger to be feared is the onset of acute pulmonary oedema. It is in the main this oedema which, in the acute stage of poisoning, threatens the life of the subject, for if abundant it causes death by asphyxiation, the patient being in fact drowned by his own exudation.

The rate of onset and the degree of oedema are dependent on the particular gas and on its concentration, and, though in some cases to a lesser degree, on the duration of exposure.

#### Phosgene Gas

30. As a type of the group, the morbid anatomy, signs and symptoms of phosgene gas poisoning will be described, after which differences due to poisoning by other gases of the group will be indicated.

31. **Morbid anatomy.**—The essential lesions are pulmonary oedema, rupture of the pulmonary alveoli, and concentration of the blood, together with thrombosis.

In one case, in which death had occurred two hours after exposure to the gas, the lungs were smaller than normal, heavy, uniformly airless and purple, and completely waterlogged. There was no emphysema. Thin serous fluid ran abundantly from the surface of the lung when it was cut across.

When death occurs in the latter part of the first day, the trachea is moderately congested, and this congestion becomes more marked in the smaller bronchial tubes. Both trachea and bronchi may be filled with a thin, yellowish, frothy and highly albuminous fluid, which may be found escaping as a white froth from the nostrils and mouth after death. Occasionally there is remarkably little inflammation in the larynx, trachea, and larger bronchi. The lungs are voluminous,

heavily œdematous, and much congested with blood. Aerated patches of emphysema, which sometimes culminate in actual bullæ, especially at the edges of the lung, alternate with depressed patches of collapse. On section, frothy serous fluid mingled with dark blood drips abundantly from the lung tissue. Air that has escaped from the lung is sometimes seen as chains of bubbles below the visceral pleura, along the interlobar fissures, and even occasionally penetrating the tissues of the mediastinum. Sometimes petechial hæmorrhages are visible on the surface of the lungs. The pleural cavities almost invariably contain a quantity of serous, perhaps bloodstained, effusion; the amount may vary from an oz. or two up to 20 oz. on each side of the chest, but it is uncertain how much of this is a post-mortem accumulation. The heart is sometimes dilated, sometimes fairly normal in volume. The veins are greatly distended with blood which often clots rapidly after death.

In the case of deaths on the second and third day the general appearance of the thoracic viscera is much the same as on the first day, the main difference being that aeration of the lung is distinctly greater, while serous fluid does not drip so freely from the cut surface of the lung. The aerated condition is greatest in the lower lobes of the lungs where they are in contact with the diaphragm, the œdema persisting longest in the upper lobe.

On the fourth day serous fluid no longer drips from the lungs on section, but commencing broncho-pneumonia and pleurisy may indicate that secondary bacterial infection has set in.

The earlier that death ensues the greater is the degree of pulmonary œdema found at post-mortem examination. The greater aeration of the lungs of cases dying on the second and third days, taken in conjunction with the clinical history of the severe cases that survive, makes it evident that the fluid is rapidly absorbed from the lungs from the second day onwards.

In severe cases a remarkable concentration of the blood may be present, the hæmoglobin percentage rising as high as 140 with a corresponding increase in the red cell count. This is brought about partly by the passage of œdema fluid into the lungs, and partly as a sequel to the condition of "shock" and want of oxygen. Associated with this concentration is the occurrence of thrombosis in the blood vessels of the lung, and also to a variable extent in those of other organs of the body.

Occasionally thrombosis takes place in the larger blood vessels, for rare instances are known in which the peripheral



arteries to the limbs have become occluded. As a rule the threatening gangrene has cleared up in these cases without amputation of the limb proving necessary.

It seems probable that the primary cause of the thrombosis in these different situations is the same, viz., damage of the vascular endothelium due to anoxæmia coupled with some slowing of the general circulation and, perhaps, with increased tendency of the blood to clot.

In some cases where death has occurred after two or three days of persistent cyanosis and unconsciousness, the white matter of the brain has been found to be peppered with tiny petechial hæmorrhages. Microscopic examination shows that the hæmorrhages are of spherical shape, with a small area of necrotic brain tissue traversed by a capillary blood vessel in the centre. They appear to be dependent on local damage of the vessel wall, associated with the formation of capillary thrombi and stasis of the blood.

Precisely similar punctate hæmorrhages have been found in severe cases of carbon monoxide poisoning, as well as in other conditions, so their occurrence cannot be regarded as a specific effect of the gas. As a general rule symptoms caused by these punctate hæmorrhages are unnoticeable owing to the extreme gravity of the patient's general condition. In one or two instances large cerebral hæmorrhages have occurred on the first or second day in cases of deep cyanosis, just as may occur in very severe and prolonged poisoning with carbon monoxide.

Petechial hæmorrhages and a slight superficial ulceration are frequently seen post mortem over the inner surface of the cardiac fundus of the stomach. How far these may owe their origin to the direct irritant effect of swallowed gas, and how far they may be dependent merely on the general venous congestion of the abdominal viscera and on the asphyxia is uncertain. In rare instances there has been serious hæmorrhage into the stomach owing to an extension of the ulcerative process.

The kidneys may be found to be enlarged and congested at autopsy, and in some instances capillary thrombi are found in the glomeruli. This change, however, rarely leads to any clinical features of renal trouble. It is very unusual for albuminuria to develop later.

**32. Symptoms and Signs.**—Casualties from poisoning by pulmonary irritants may be classified, according to their modes of onset, into two chief groups: Acute, with violent onset, and Acute, with insidious onset.

**33. *Acute with violent onset.***—Exposure to an atmosphere

containing phosgene causes immediate sensory irritation of the respiratory passages accompanied by smarting and watering of the eyes. This irritation of the respiratory passages causes catching of the breath, coughing and a sensation of tightness and constriction and pain in the chest. After the initial check, the breathing continues, but is gasping in character and interrupted by violent fits of coughing. After getting out of the poisonous atmosphere the respiration remains rapid and shallow, any attempt to draw a deep breath giving rise to painful discomfort and provoking a fit of coughing. Nausea, retching, and vomiting are prominent features in the early stages of poisoning. There is slight or profuse expectoration. Headache, and a sense of fatigue in all the limbs, often prostrate the patient.

As œdema develops in the lungs, the breathing becomes rapid and panting, but of a characteristically shallow type, unlike the deep ventilation of a healthy man after running, and more resembling the fast respiration of a child with broncho-pneumonia. The ears, lips and progressively the entire face assume a cyanotic, bluish-red tint which may deepen to the intense violet of fullest cyanosis, and there may be visible distension of the superficial veins of the face, neck or chest—especially in persons gassed with pure chlorine. In phosgene poisoning this full cyanosis is often omitted, and the patient passes rapidly into a state of circulatory collapse, with a feeble, flickering pulse of over 120, a cold clammy skin, and a leaden hue in the face, in which only the lips and tips of the ears reveal the asphyxial cyanosis that underlies the failure of the man to win his fight for life. While in the stage of cyanosis, whether “blue” or “grey,” the patient is always restless and very apprehensive of the seriousness of his condition. The expression is anxious and distressed, with the eyeballs staring and the lids half closed. At this stage casualties can be divided into three types :—

- (a) The milder case, with reddish flush in the face, with some hurry of respiration, and with pain in the chest and epigastrium which is increased by coughing.
- (b) The severe case with “blue” cyanosed face, distended neck veins, and full, strong pulse of 100.
- (c) The severely collapsed case, with leaden “grey” cyanosis of the face, and rapid, thready pulse.

The milder case is often drowsy and soon falls into a sleep from which he wakes refreshed. Coughing upon a deep breath, occasional vomiting after food or drink and a slight sense of rawness in the throat together with general debility, may persist for a few days after which the patient becomes



convalescent. During the early days of convalescence there is often a considerable slowing of the pulse from vagus action, which may bring it down to about 50 or even 45 in the minute. Such early bradycardia is often seen also in recovery from severer poisoning; it has no serious import, but is rather a sign that the patient is beginning to convalesce.

Cases of severer cyanosis, if the depth of the reddish-blue colour is well maintained and the pulse does not exceed 100, tend to recover in two or three days, and their recovery is generally similar to that of the milder cases. Provided that the circulation and the activity of the respiratory centre can be maintained, the œdematous fluid in the lungs is soon absorbed, most of it vanishing by the fourth or fifth day. At any time, however, particularly if subjected to much physical effort, those cases may rapidly pass into the most dangerous condition of "grey" cyanosis and collapse. The pulse becomes rapid, thready and irregular. The patient, though obviously weaker, becomes more restless and slightly wandering in mind, or semi-comatose.

Even the worst of the "grey" cases may recover with proper treatment, but the mortality among them is always distressingly high. Recovery from this state of depressed circulation may be succeeded by severe and even fatal bronchopneumonia. When this infective complication develops, the sputum becomes purulent and the temperature rises. Death usually follows rapidly. If the case lasts into the third week after gassing, he may justly be expected to survive the acute infection.

In 81 per cent of deaths due to poisoning by phosgene and chlorine the death occurred within 24 hours.

34. *Acute cases with insidious onset.*—Cases have not infrequently been reported in which men who have been exposed to gas have been able to carry on their work for an hour or two with only trivial discomfort, and even to march from the trenches to their billets, and then have become rapidly worse, and passed into a condition of collapse with progressive œdema of the lungs that may prove rapidly fatal. In such cases the ingestion of a heavy meal seems sometimes to have had a prejudicial effect. At other times men who have passed through a gas attack and have subsequently complained of only slight cough, nausea, and tightness of the chest whilst resting in the trenches, have collapsed and even died abruptly some hours later on attempting to perform some vigorous muscular effort. A minor degree of the same effect is sometimes shown when men who have been slightly gassed find on trying to walk down from the trenches that they get unusually "done in" and breathless, and are obliged to rest frequently.

In these cases the deficiency of oxygen, the result probably of pulmonary oedema already existing, has not been felt until muscular exertion increased the need for oxygen.

35. One very striking example of this delayed effect may be cited in which the patient was observed from start to finish after only a brief exposure to a strong concentration of phosgene. The greatest care was taken to prevent any muscular exertion, so that no complicating factor was introduced. The immediate irritant symptoms and coughing that were produced during the exposure soon diminished in fresh air, and an hour and a half later there was no coughing and the patient seemed practically well, the pulse being normal. The condition remained quite good till four and a half hours after exposure to the gas, when the patient became bluish about the lips. Coughing then recommenced with the expectoration of frothy sputum. Soon the lips and face became of a grey ashen colour, though the pulse remained fairly strong. About four pints of clear, frothy, yellowish liquid were coughed up from the lungs in the next hour and a quarter, and at the end of this time the patient expired. At no time was there any great struggle for breath, nor did the patient realize at all how ill he was.

36. **Physical signs.**—The percussion note may remain resonant over the chest, notwithstanding the existence of marked pulmonary oedema. In many cases, however, the note is impaired, especially over the back. The breath sounds are weakened, especially behind ; they may also be harsh in character, but are not otherwise altered, and are never tubular. Fine rales are heard, more especially in the axillary region, and the back and sides of the chest. Rhonchi are also occasionally heard. With the development of inflammatory complications the physical signs become those of pleurisy, bronchitis, or broncho-pneumonia.

In the early acute stage, however, the physical signs on examination of the chest give little indication of the very serious extent to which the lungs may be damaged. The colour, the pulse, and the character of the respiration are the chief guides to prognosis.

37. **Symptoms during convalescence.**—Poisoning by the lung irritant gases is sometimes followed by recurring frontal headache, epigastric pain, which is often worse after food but is rarely severe and rapidly disappears, mild bronchitis, prolonged inability for severe muscular work or even for moderate exercise and associated with præcordial pain, tachycardia and a rapid, shallow type of breathing. Spas-



modic attacks of nocturnal "asthma" sometimes occur. These recur at varying intervals and last from three to thirty minutes. In uncomplicated cases unassociated with disordered action of the heart, exercise by day does not cause dyspnoea nor any tendency to increase of nocturnal asthma, but after the exercise there may be intense headache and giddiness and often an abnormally slow pulse. In these cases it has been found that the hæmoglobin percentage is always increased, up to 110 or 120 per cent, whereas this change is not found in pure disordered action of the heart after gassing. The red cells are correspondingly increased, a case for example having been recorded with 6,900,000 a cubic millimetre, with hæmoglobin 116 per cent, and a normal colour index.

38. It has been found that a fair proportion of the ordinary cases of dyspnoea and tachycardia can soon be hardened by carefully regulated exercises. There are, however, no satisfactory criteria for an early estimate of when recovery will be, except the way in which the pulse and patient react to exercise. The majority, after a period of hardening at a convalescent depot, should be fit to rejoin their units within three months.

39. In some instances, partly drawn from severe cases of pulmonary oedema and partly from others less severely gassed, the features of short-windedness and disordered action of the heart persist and are undoubtedly the result of gassing alone. Professor Barcroft and his colleagues at Cambridge have confirmed the observation that some of these cases showed a persistently high red cell count, that is, a true polycythæmia without reduction of the blood volume, and not the transient relative increase seen immediately after gassing. The condition could be reproduced in rabbits after a brief exposure to phosgene or chlorine, when there was first the early concentration of the blood, and after that a steadily developing and persistent increase in the red cell count. Rabbits with this condition, two months after gassing, had their count restored to normal by life in a chamber containing 40 to 50 per cent of oxygen, or twice the proportion in ordinary air. Similarly good results were obtained when gas casualties with disordered action of the heart and polycythæmia were placed for five days in a chamber containing 40 to 50 per cent of oxygen. In the case of rabbits it was proved microscopically that the respiratory surfaces of the lungs had been damaged by a thickening of the pulmonary epithelium and by a bronchiolitis obliterans. No corresponding examination has been made of the lungs of gas casualties at a late date after exposure. But the close comparison of this condition with that seen in

men living for a long time at high altitudes under conditions of oxygen shortage, and the fact that these gas patients were temporarily improved by treatment in the chamber enriched with oxygen, did suggest that the condition was due to a persistent anoxæmia, indirectly weakening the heart, and perhaps itself caused by injury to the lungs.

40. Professor Haldane, at Oxford, developed a different view. This laid emphasis on the rapid shallow type of breathing, and inability to hold the breath for long, that characterized these men. Rapid, shallow breathing, say at 50 a minute, does not adequately ventilate the lungs. In a healthy man it will, if deliberately maintained, eventually lead to giddiness, faintness, and various phenomena of oxygen shortage. In his view the anoxæmia and the consequent debility and circulatory deficiencies might be caused simply by the neurasthenic persistence of the rapid shallow breathing that had first been established when the man was poisoned by gas, brought about through changes in the sensitiveness of the Hering-Breuer reflex in the medulla that depends upon afferent vagal stimuli from the lungs.

41. Whatever the evidence for anoxæmia may be, and whatever the deeper physiological explanation of this state, the certain fact was that a small proportion of gas casualties did develop an intractable form of disordered action of the heart, and that the tendency to this invalidism was increased if the men were pressed to physical effort too early and too fast at the beginning of convalescence. Some of these men showed polycythæmia, others did not. It is almost certain that the disordered cardiovascular state did not have its pathological origin simply in the mechanical strain thrown on the circulation by the transitory asphyxial congestion after gassing. Some clinicians were inclined to see in it further evidence of a toxic action of phosgene on the muscle of the heart and blood vessels, akin to that which was originally assumed in explanation of cardiac collapse and death in the acute stages of poisoning. But physiological analysis was always opposed to the acceptance of this view either for the acute cases or for the chronic invalids.

42. There is no evidence that pulmonary tubercle or any other serious maladies have developed more frequently in individuals poisoned by pulmonary irritants than among any other classes of pensioners.



## Chlorine

43. A much stronger concentration of this gas is needed to cause severe pulmonary œdema, or even lachrymation, than is the case with phosgene. It is, however, far more irritant to the respiratory passages than is phosgene. A very marked feature in the early attacks, when chlorine alone was used, was the paroxysmal and violent coughing, which not only occurred during the exposure but also persisted for a long time afterwards. Emphysematous changes were pronounced, and subcutaneous emphysema of the neck and chest occurred in a number of instances. As a general rule the cases exhibited deep cyanosis rather than pallor and collapse, with a fairly full pulse and much dyspnœa. Copious frothy expectoration was common.

44. Delay in the onset of serious symptoms is not evident in chlorine poisoning. Though exudation of fluid into the lungs may not perhaps start at once, the violent paroxysms of coughing, the painful dyspnœa, and the repeated attacks of vomiting convey the impression that the case is severely ill from the start.

## Chloromethyl Chloroformate and Trichloromethyl Chloroformate

45. Both these gases give rise to effects similar to those produced by phosgene. Trichloromethyl chloroformate has about the same toxicity as phosgene, but greater lachrymatory power; chloromethyl chloroformate has a toxicity of the same order as that of chlorine.

## Chloropicrin

46. This is a stronger lachrymatory agent than is trichloromethyl chloroformate, though it is a good deal inferior in this respect to the true lachrymators. For such lengths of exposure as are likely to occur in the field, chloropicrin must be at a distinctly higher concentration than phosgene to cause severe pulmonary œdema, but it is much more deadly than chlorine. It causes greater sensory irritation of the respiratory passages than does chlorine. Pain in the chest and epigastrium, abdominal discomfort, and violent attacks of vomiting are exceptionally marked. Brief exposure to strong concentrations may cause temporary unconsciousness.

The substance is semi-persistent and the gas may remain in considerable concentrations in the neighbourhood of a shell burst for as long as 12 hours.

47. Unlike phosgene, chloropicrin is a substance of considerable chemical stability when in contact with animal tissues, and it is cumulative in action, for experiments on animals have shown that prolonged exposure to very low concentrations may still give rise to serious toxic symptoms, while equally low concentrations of chlorine or phosgene may be quite ineffective.

48. Frequent exposure to small doses of chloropicrin, which would have only a trivial effect in themselves, may gradually lead to a greatly increased susceptibility to the gas. A man who has acquired this susceptibility is liable to attacks of "asthma" whenever he has been exposed to a trace of chloropicrin in the air. The attacks usually occur at night, and are characterized by the sudden onset of a rapid, shallow type of breathing associated with a feeling of tightness of the chest and a sensation of suffocation, which causes considerable distress. There is usually a short, dry cough at intervals, which is occasionally followed by the expectoration of a small quantity of tenacious mucus. The attacks may last for as long as a couple of hours, and appear to resemble, though in exaggerated degree, the attacks of "nocturnal dyspnoea" which have already been described as occurring during convalescence from phosgene poisoning.

49. Chloropicrin has also an irritant action externally. The liquid applied to the skin may cause erythema or even ulceration, and such lesions are prone to become septic. Chloropicrin vapour may cause conjunctivitis and the liquid splashed on the cornea may give rise to ulceration.

50. Chlorine, chloropicrin and the chloromethyl and trichloromethyl chloroformate cause much more damage to the mucous membrane of the respiratory passages than does phosgene.

### **Phenyl Carbylamine Chloride**

51. This substance has a very offensive mustard-like smell, and is very liable to cause immediate nausea when inhaled. It is a lung irritant of only moderate power, and is apt to cause bronchitis. In addition, it is a moderate lachrymator.

### **Physiological Factors in relation to Symptoms and Treatment**

52. Severe cases of poisoning by the lung irritant gases fall, as has been pointed out, mainly into two groups. The first of these is characterized by intense florid and deep cyanosis with vascular congestion and engorgement of the veins,



while the second exhibits no venous engorgement but pallor, like that seen in collapse, with leaden-grey or lilac-coloured lips and tongue. The common feature in the two groups is the cyanosis, whether of the florid and congestive or the grey type, for it indicates that in both cases the patient is suffering from profound shortage of oxygen, and it is this shortage of oxygen that endangers life. Striking though the appearance of the first type may be, the grey or pallid type is clinically of far more serious import.

In the case of concentrations of the gas which are met with in practice in the field the whole of the irritant gas appears to be stopped by the tissues of the lungs, none penetrating as such into the blood stream, though alteration of the blood pigment shows that in experimental exposure to exceedingly high concentrations of the gas such a penetration is possible. But in bearing the brunt of the attack the respiratory organs suffer severe damage, and the asphyxial symptoms must be attributed to this damage.

All the lung irritant gases cause severe inflammatory changes in the pulmonary alveoli associated with widespread pulmonary oedema. It is easy to see that swelling of the alveolar epithelium and the interposition of a layer of oedema fluid must seriously interfere with the gaseous exchange between the blood and the air in the lungs, the difficulty being increased by thrombosis in some of the damaged pulmonary capillaries, a feature always exhibited to a greater or less degree. It must, however, be remembered that  $\text{CO}_2$  is a far more soluble gas than oxygen, and on this account will diffuse through a layer of liquid at a far higher proportionate rate than oxygen. A case can, therefore, be imagined in which the accumulation of oedema fluid in the pulmonary alveoli may seriously reduce the rate at which oxygen can gain admission to the blood whilst there is little or no material impediment to the passage of  $\text{CO}_2$  in the reverse direction. A typical case of severe phosgene poisoning appears to offer an example of this type, a picture, indeed, of gross deprivation of oxygen unaccompanied by any damming back of  $\text{CO}_2$  in the body. As the oedema develops—very often insidiously, though in the end rapidly—cyanosis appears, and soon both nervous system and heart begin to feel the effects of the shortage of oxygen. In the absence of the stimulus which might be afforded by accumulation of  $\text{CO}_2$ , the respiratory centre begins to fail and the heart to weaken, and commencing failure of the respiration and circulation merely enhance in their turn the shortage of oxygen supply to the tissues which is primarily dependent on the morbid changes in the lungs. A vicious circle has been established, and the condition of the patient can but pass from bad to worse unless

this circle can be broken, for the typical features of collapse will soon become evident in the rapid and shallow respirations, the feeble, rapid and flickering pulse, the cold and clammy skin and the leaden-hued pallor.

Exposure to an atmosphere containing phosgene in sufficient amount to cause in the end a fatal pulmonary oedema may be accompanied by relatively little sensory irritation of the upper respiratory passages, and coughing may be inconspicuous, but the case is different with such a gas as chlorine, which not only causes far greater sensory irritation of the upper respiratory passages and provokes vigorous coughing, but also causes far more obvious inflammation of the epithelium of the bronchial tubes and spasm than does phosgene. The violent respiratory movements resulting from the coughing and efforts to draw breath, when many of the bronchial tubes are getting blocked by inflammatory exudate or muscular constriction, tend to tear the damaged lung tissue so that areas of emphysema with surrounding areas of collapsed lung are formed. Such disorganization of the lung structure implies an interference with the normal ventilation of the lung, for the collapsed areas at least will be inadequately supplied with air, and this cannot be compensated by over ventilation of the emphysematous parts where the proportion of sound alveolar wall to air will be unduly low. Interference with the proper ventilation of the lungs must impede the elimination of  $\text{CO}_2$  from the body as well as the due absorption of oxygen, and this effect will be superimposed on that caused by the accumulation of oedema fluid in the alveoli. The case will now exhibit the features of serious shortage of oxygen accompanied by retention of  $\text{CO}_2$ .  $\text{CO}_2$  is, however, a natural stimulus to the respiratory centre, and its presence in excess, when there is deficiency of oxygen as well, will help to maintain the action of the heart and the tone of the circulation. We shall find, therefore, that the breathing movements will be far more vigorous and effective than in the case suffering from simple deficiency of oxygen, which exhibits the rapid and shallow type of respiration characteristic of failure of the respiratory centre, and that though deep cyanosis will indicate great shortage of oxygen in the blood the heart will be beating at a slower rate, and far more steadily and powerfully. The obvious venous congestion shows that the heart is, however, definitely feeling the shortage of oxygen and is unable to cope adequately with the strain it has to bear. The pathological changes in the lungs must offer an obstacle to the pulmonary circulation and the circulation as a whole has to contend with yet another difficulty—concentration and increased viscosity of the blood, which is shown by a definite and even considerable increase in the red



cell count and hæmoglobin percentage. This increase in the concentration of the blood is dependent in part on the loss of fluid into the lungs, and in part on the passage of plasma from the capillaries of the body into the tissue spaces, a phenomenon observed in other conditions of "shock" accompanied by failing circulation.

The rapid and shallow type of breathing characteristic of failure of the respiratory centre owing to gross lack of oxygen has been described; but we may also see a rapid and, perhaps, shallow type of breathing at an early stage of poisoning by the lung irritant gases before any material degree of œdema has developed. This is apparently due to irritation of the vagus nerve-endings in the lungs by the gas. Afferent impulses passing up branches of the vagus nerves connected with the lungs are concerned, in normal respiration, with the switching of the inspiratory phase of the breathing movements into the expiratory phase at the correct moment and vice versa, and irritation of these nerve-endings quickens the rhythm of breathing. This condition can be observed in animals exposed to an irritant gas, and the quick rhythm is abolished by section of the vagus nerves.

The above considerations suggest some possible lines of treatment. As the danger to life is primarily due to the hindrance of the passage of oxygen from the air in the lungs into the blood, we may attempt to compensate for this by increasing the concentration of oxygen in the lungs by the administration of oxygen so as to increase the speed of diffusion of gas through the œdema fluid. If there is a shortage of oxygen when the patient is at rest muscular exertion, which causes the body's demand for oxygen to increase greatly, can do nothing but harm, and exposure to cold, since it results in increased metabolism, will have a similar bad effect. The adoption of a suitable posture may mechanically assist the expulsion of œdema fluid from the lungs. In the cases of congestive cyanosis where we suspect  $\text{CO}_2$  retention to be an added factor, we can see that the  $\text{CO}_2$  retention is actually beneficial and so need not worry about it, at all events in the first instance. Marked venous congestion will suggest the possibility of venesection with a view to giving relief to an overloaded heart at a critical time. The concentration of the blood makes it worth while considering whether intravenous infusion of saline may not be beneficial, as it is in other cases of shock.

## The Treatment of Poisoning caused by the Lung Irritant Gases

53. Cases of all degrees of severity may occur, and sometimes it is not easy for the medical officer to decide whether or not a man has really been gassed. The fact that delayed action is liable to be exhibited by the lung irritants introduces another factor of uncertainty.

In these doubtful cases the patient should be made to give his own account of the occurrence, in order to see whether it suggests that he may have been exposed to a significant dose of gas. He should be allowed to describe his own symptoms. No leading questions should be put to him. Some definite objective symptoms, such as vomiting, may have occurred. He may have been unduly exhausted by trying to walk to the aid post, and have had to be helped down ; evidence in support of this may possibly be furnished by the pallor of his face and by a rapid pulse. Careful examination should be made to see if there is any lachrymation, any sign of cough, or unduly short and rapid breathing. It should be ascertained whether he can take a deep breath without affording any evidence that this gives rise to discomfort or provokes a cough.

The benefit of the doubt must be given to the patient, but it should be borne in mind that if no objective symptoms have arisen after the lapse of 48 hours, the degree of gassing must have been very slight, and the case can be returned to duty with little delay.

### *Treatment in the acute stage*

54. *Rest.*—The importance of rest cannot be exaggerated. In the earlier stages undue muscular exertion is liable to lead to great aggravation of the symptoms. When pulmonary œdema is well established, and the respiratory exchange in the lungs is seriously interfered with, it is of importance to keep the oxygen consumption as low as possible, and activity of the muscles is by far the most potent cause of increased oxygen consumption.

55. All gas cases should be evacuated as soon as possible to the point at which arrangements have been made for effective treatment (a Field Ambulance or a Casualty Clearing Station, according to circumstances). Whilst waiting for evacuation care should be taken that the cases do not undergo muscular exertion. All except the lightest cases should as far as possible be evacuated lying down, and walking cases should be given every assistance whilst going back along the trenches, so that they may avoid physical effort as much as possible. Walking



cases should not be allowed to carry their equipment. If any walking case shows signs of increased severity in his symptoms (marked breathlessness, palpitation, loss of power in his limbs, or a feeling of being "done in") arrangements should at once be made to carry him. Care should be taken that the breathing of any cases showing material symptoms is not impeded by tight belts or braces.

56. When cases reach the point where they can be retained for treatment they should be kept lying down until any obvious symptoms due to the gassing have subsided. Those who show definite symptoms should not be allowed to leave their bed or stretcher for any purpose whatever.

57. Uniform should be exchanged for hospital clothing wherever practicable. The pure pulmonary irritants do not adhere for long to clothing during exposure, but the lachrymators, the arsenic substances and mustard do so, and the general rule should therefore be observed with all casualties. If this is not done the patient is exposed to a longer action of the poison and moreover the hospital staff may be affected. Orderlies engaged in this task should wear respirators and protective clothing.

58. *Warmth*.—The greatest care should be taken to keep the cases warm, and attention must be particularly directed to this point if any of the clothing has to be removed. Warmth will not only help to combat shock, but will diminish any tendency to the muscular movements of shivering.

59. *Oxygen*.—As deficiency of oxygen is an essential pathological result of poisoning by a lung irritant, treatment must be fundamentally devoted to supplying the deficiency by administration of oxygen. The aim should be to tide the case over the critical period of the first two days.

Oxygen should always be given to casualties with serious pulmonary œdema, that is, to men with intense blue cyanosis or grey pallor. These need oxygen continuously and over a long period. If the supply permits of such use, it should be given also to milder cases of œdema in order to prevent their lapsing into a more serious state of asphyxia. It should be remembered that in every autopsy following early death from pulmonary irritant poisoning, extreme œdema of the lungs was found, that cyanosis is the main indication of such œdema, and that no case in whom it was possible to restore a pink colour by the proper use of oxygen died from simple pulmonary œdema. The oxygen, which does not require to be warmed, must be given with some special apparatus, such as a Haldane mask or nasal catheter, that will ensure its being adequately

commingled with the respired air. The minimal current of oxygen should be used, from two up to even ten litres enrichment a minute, which will suffice to keep the patient's face pink, a change that is usually associated with improvement of the pulse; and the treatment must be maintained, even for one or two days and nights with progressive lessening of the oxygen supply, until recovery is assured and the patient does not lapse into cyanosis when the oxygen is intermitted. If no other apparatus is available, then the nitrous oxide anæsthetic apparatus, which delivers pure oxygen from the cylinder (the valve of the bag being, of course, set so that the patient expires into the air and does not breathe in and out of the bag), should be used intermittently, say at alternating intervals of five minutes, because pure oxygen may eventually be irritant to the lungs.

The Haldane oxygen apparatus allows of continuous administration of oxygen at any required concentration over long periods. It is designed so that oxygen only passes to the face mask during inspiration, the oxygen being therefore used with the greatest possible economy. In practice the oxygen supply is increased until the patient's face becomes of a normal colour, and then the supply is kept constant at this level. Relief of the cyanosis shows that the hæmoglobin in the blood is properly oxygenated, and no additional advantage will be obtained by increasing the oxygen supply further.

When oxygen is given continuously for prolonged periods by whatever method, intermission should be made for five minutes at the end of each half hour.

If the Haldane oxygen apparatus is not available, or the patient, as sometimes happens, refuses to tolerate the mask, the best method to adopt is to administer oxygen through a nasal tube. A soft rubber catheter should be lubricated and gently introduced into the nostril until its extremity lies in the naso pharynx; it can be secured in position by adhesive plaster. A continuous stream of oxygen is allowed to pass from a cylinder through the nasal tube. This method is more wasteful of oxygen than is the Haldane apparatus, since oxygen continues to pass during expiration, and is lost to the patient at this time, and consequently a far greater rate of flow of oxygen is necessary to get a given result with the nasal tube than with the Haldane apparatus.

Colonel Stokes devised a distributing apparatus by which many men could be simultaneously treated with oxygen. A lead from the oxygen cylinder was taken to an oil drum, which acted as a pressure reducing chamber, and from this several rubber leads branched off to nozzles which could be taken to the bedside, each being controlled by a stopcock.



Respirator facepieces may be used as masks for the supply of oxygen to patients.

Subcutaneous injection of oxygen has proved valueless; neither have efforts to introduce oxygen intravenously met with any success.

The oxygen frequently gives obvious relief to the general condition of the patients. They become less anxious and restless, so that they are sometimes unwilling to have the administration interrupted when they have once got used to the apparatus. At first they may be very unwilling to tolerate any method of administration, and every effort should be made to gain their confidence.

If panting is present, this usually persists even when the cyanosis has been relieved, since it is mainly due to retention of carbonic acid. Sometimes in very severe cases administration of oxygen increases the panting. This is probably due to the fact that the respiratory centre itself has begun to fail from want of oxygen, and is responding inadequately to the natural stimulus of carbon dioxide. The want of oxygen having been relieved, the respiratory centre regains its normal irritability and reacts fully, but hyperpnœa will be exaggerated until the carbon dioxide which has accumulated in the body owing to the relatively deficient breathing has been washed out by the increased respiration. If it is feared that such hyperpnœa may in itself do harm to the patient, oxygen should be given intermittently for a time, and continuous administration can be started later on.

60. *Venesection*.—There is no doubt that cases of deep cyanosis with a full pulse and signs of venous engorgement may be greatly benefited by a venesection up to 20 ounces, the blood being withdrawn slowly. The headache often disappears, dyspnœa is somewhat diminished and sleep follows. By this procedure relief may be afforded at a critical period when the right side of the heart is beginning to give way under the strain.

Venesection is contra-indicated in those cases which show pallor and collapse with a very rapid, thready pulse.

Apart from its effect on the circulation, venesection has been advocated in cases of phosgene poisoning as a measure likely to diminish the amount of fluid exuded into the lungs. Early venesection as soon as pulmonary œdema is recognized has been recommended by medical officers attached to the South African mines in cases of poisoning with nitrous fumes, and there was an impression amongst medical officers at the Front, and this was supported by French opinion, that similar early venesection is beneficial in cases of phosgene poisoning and the like. Experiments made on animals late in the War

indicated that when venesection was combined with the intravenous infusion of isotonic salt solution, still better survival results might be expected. No evidence appeared in the experiments to suggest that infusion, when performed some time after the venesection, led to increase of lung oedema. This was not applied to human casualties, but it does appear that the method might prove a useful means of treatment in cases with pulmonary oedema where hæmoglobin estimation shows an unduly high concentration of the blood.

### *Drugs*

61. *Atropine* has been used under the impression that it will relieve bronchial spasm and check the output of oedema fluid. There is, however, no clear evidence that it has any beneficial action in practice, and as it may lead to acceleration of the heart it should not be used.

62. *Cardiac Stimulants*.—On the whole brandy has proved one of the most effective stimulants. Pituitrin 0·5 c.c. hypodermically at intervals of not less than three hours has certainly been of value in some serious cases. Hypodermic injection of camphor (*e.g.* camphor gr. 1, olive oil min. 5, ether min. 5, minims 10–20 for a dose), or of caffeine (*e.g.* caffeine gr. 10, sodium salicylate gr. 17, distilled water to one drachm, minims 10 for a dose) have also proved of value. Neither digitalis nor strychnine have shown themselves of much use.

Oxygen is far the best cardiac stimulant. When once oxygen treatment has been started, and the major disadvantage under which the heart is labouring has been removed, the cardiac stimulant drugs may have a valuable additional effect.

63. *Morphia* is a dangerous drug to use when the respiration is seriously affected. Its use should therefore be restricted to severe cases where restlessness is extreme, and can be controlled in no other way. The dose should be small, gr.  $\frac{1}{8}$ , or 15–20 minims of tinct. opii.

64. *Expectorants*.—These should not be given to severe cases during the first two days for fear of increasing the tendency to cough, and so augmenting the damage in the lungs. An ordinary expectorant mixture containing ammon. carb. and vin. ipecac. may with advantage be given to the milder cases, as well as to severe cases when the acute symptoms have definitely begun to abate.

65. *Phenacetin* should not be used for the relief of the headache that usually occurs; it is somewhat liable to bring



on collapse, though probably the same objection does not apply to aspirin. Relief of oxygen-want is the best means of relieving headache.

### Methods of Aiding the Discharge of Exudate from the Lungs

66. *Vomiting*.—Repeated vomiting frequently occurs spontaneously in cases of poisoning with the acute lung irritants. In the earlier stages of acute pulmonary oedema this may prove of considerable benefit in promoting the discharge of fluid from the lungs. If vomiting does not occur naturally, it may be encouraged at this stage by simple measures, *e.g.* salt and water and tickling the back of the throat, but neither apomorphine nor ipecacuanha should be used for this purpose because of the great depression that they produce subsequently. As the effort of vomiting is very exhausting, it should not be encouraged when the patient at a later stage begins to fail.

67. *Posture*.—If much thin fluid is being expectorated, good results are sometimes obtained by turning the head of the patient sideways and raising the foot of the bed or stretcher three or four feet, or even higher, for a few minutes at a time, with the idea of draining fluid from the chest.

68. *Schäfer's artificial respiration* has occasionally proved of service in expelling fluid from the chest, but it is necessary to watch the effect on the patient very closely lest disaster ensue owing to a large amount of fluid being suddenly forced into the bronchial tubes.

### General Treatment

69. Open-air treatment is good under favourable conditions of weather, but the slight gain from a fresher atmosphere is more than counterbalanced by the difficulties of nursing the patients and of keeping them warm so as to counteract the effects of shock. It is therefore preferable to treat at least the more severe cases in a well-ventilated ward. In bad weather tents and marquees are apt to be very dark, which makes it difficult to appreciate the degree of cyanosis shown by the patients.

Bad cases should if possible be put to bed, rather than left on stretchers. The cases should be allowed to assume the posture that they find most comfortable; some prefer to lie down, others to be propped up.

Those with broncho-pneumonia should be segregated by at least six feet, if they cannot be treated in a separate ward.

Food should only be given in fluid form and sparingly in the acute stage, and the diet should be kept light until the patient is definitely convalescent. Pain in the epigastrium sometimes yields to small doses of sodium bicarbonate. Measures should be taken to keep the bowels open.

Severe cases complain greatly of thirst in the acute stage. There appears to be no valid reason for withholding fluids, and water (brandy may be added if desired), or tea may be given; the fluids should, however, be given in small quantities at a time.

The mouth usually becomes dry and foul, and attention should be paid to this, as it is a source of great discomfort to the patient.

So far as circumstances permit, no case should be evacuated to the L. of C. until definite cyanosis or serious symptoms have disappeared. It is most important that a note should be made on the Field Medical Card of the symptoms of those who have passed through a condition of gravity for the guidance of the medical officer on the L. of C. in his disposal of the cases.

70. *Treatment during the convalescence stage.*—Most of the milder casualties sent to the L. of C. are likely to become fit for duty after a short rest. Bronchitis and gastric disturbance as a rule subside quickly with ordinary treatment. Patients who develop secondary broncho-pneumonia, and the rare cases showing dilation of the heart, will naturally be regarded as serious and treated accordingly. All those who pass through an acute stage of cyanosis must be regarded as serious and given a prolonged period of convalescence. The cases which show tachycardia and dyspnoeic symptoms offer a problem of much greater difficulty. In practically all these cases examination of the chest reveals no obvious physical signs to account for the symptoms, and the cases usually look surprisingly well while they are resting, though the occurrence of attacks of nocturnal dyspnoea may call attention to the fact that the case is definitely abnormal.

In order to differentiate these cases recourse may be had to the reaction after exercise. The ultimate standard of a man's fitness for duty must be his capacity to perform muscular exercise without undue exhaustion, tachycardia, or breathlessness. The effect of exercise ought to be tried on all cases before they are sent from hospital or the convalescent camp to duty.

71. Cases of moderate severity which have been confined to bed for some days require testing carefully in this respect. Some such method as the following may be employed :—After



the patient has been allowed out of bed for four days he is made to walk half a mile at a steady and moderate pace. If the panting and pulse rate diminish with normal rapidity after stopping the walk, a walk of one mile is taken the next day, and if this is not too much, a walk of three miles without stopping is taken on the following day. If, however, the test exercise results in undue tachycardia and breathlessness, the best method of treatment to adopt is a course of carefully graduated exercise, and the greatest care must be taken not to overdo this exercise at the start.

72. The mildest cases, who are perhaps only retained in a medical unit for a day or two (as well as other convalescents who are well on the way to recovery), should be allowed the opportunity of resting lying down during the daytime, if they desire to do so, so as to prevent them from unduly exhausting themselves and possibly doing themselves harm.

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## CHAPTER IV

### SENSORY IRRITANTS

#### The Arsenical Warfare Gases

73. All the arsenical poisons in use as chemical warfare agents may be regarded as derivatives of arseniuretted hydrogen (arsine  $\text{AsH}_3$ ), the hydrogen atoms being replaced by organic groups such as phenyl ( $\text{C}_6\text{H}_5$ ) and atoms or groups such as chlorine (Cl) or cyanogen (CN). This change to a large extent masks the ordinary poisonous properties of arsenic, the compounds, even when present in extremely minute concentrations, showing an immediate and intense power of irritating certain sensory nerves, and it is this property which renders them valuable for military purposes. When an atmosphere containing these compounds is breathed, they do not cause gastro-enteritis, or hæmolytic jaundice or chronic peripheral neuritis. Many such arsenical compounds have been produced but at present there are only three which are likely to be met with in the field :

- (1) Diphenyl-chloroarsine.
- (2) Diphenylamine-chloroarsine.
- (3) Diphenyl-cyanoarsine.

74. Those arsines commonly used have a very high boiling point and it is difficult to get more than an extremely low

percentage in the air. They are, however, readily dispersed without decomposition by the aid of heat, in the form of an intensely irritant smoke, *i.e.* in actual particles of an extremely minute size. A substance in this very fine particulate form can remain suspended in the air, and the effects produced during the Great War depended on the liberation of part at least of the poison as a particulate cloud by the heat and violence of the explosion of a shell.

Such irritant smokes can also be liberated from suitable generators.

75. The sensory irritant poisons are effective in much lower concentrations than any of the other chemical warfare agents. In very low concentrations, after a short period of delay they cause slight and transitory nasal irritation. In higher concentrations the irritation shows itself sooner and in rapidly increasing severity. On withdrawing from the affected atmosphere, the symptoms, unlike those caused by the lachrymatory group of substances, do not immediately diminish, but tend rather to increase in severity before commencing to subside.

These substances, being particulate clouds, are not stopped by the charcoal of the respirator, but for protection a special filter is necessary. They were introduced by the Germans partly in the belief that minute irritant particles would penetrate the container of the respirator, cause embarrassment of breathing, and induce the individual to discard his mask, and thereby render him a casualty by a more lethal gas such as phosgene. Shell containing these substances were, therefore, often fired along with those containing asphyxiant gases.

Towards the end of the war shell containing these substances were used in increasing number by the Germans, but in spite of the valuable effects attributed to them, they were, taking them all round, ineffective in the field. The reason for this is attributed to the fact that the method of dispersion was faulty. Just before the Armistice a more efficient method of dispersing toxic smokes by means of generators was introduced. Generators require no special precautions for storage, are easily handled and functioned by infantry, and therefore, the ease with which toxic smokes can be employed when an attack is launched, makes the sensory irritants an important group of chemical warfare agents.

76. **Morbid anatomy.**—No fatal case of poisoning by these compounds occurred in our Army in France, so there are no data available as to the anatomical changes occurring



in man. Under appropriate experimental conditions with higher concentrations in the laboratory it has been shown that the substance is capable of exerting a powerful lethal effect on animals, death being due to pulmonary oedema and pneumonia accompanied by destructive inflammation of the trachea and bronchi and a marked fibrinous bronchitis. Some of these animals die within a few hours of exposure, and death in such cases may be largely due to shock. In those dying later, it is due chiefly to want of oxygen following the pulmonary lesions. Arsenic in small quantities can be recovered from the blood and urine of such cases.

**77. Signs and Symptoms.**—The symptoms are immediate in onset and are quite characteristic; minute concentrations in the air cause burning pain in the nose, mouth and throat, pain in the gums and jaws, repeated sneezing, a burning sensation of the face, aching pain in the eyes and frontal headache, watering of the eyes and painful conjunctivitis, copious watery discharge from the nose, tightness and burning pain in the chest, salivation, pain in the stomach, nausea, retching and sometimes vomiting which in some cases is followed by tenesmus, though diarrhoea is practically never met with. A further characteristic of the sensory irritation caused by these compounds is that the symptoms do not diminish as soon as the person affected leaves the poisonous atmosphere, but actually increase in severity for a time before they begin to subside. It may take from  $\frac{1}{2}$  to 1 or 2 hours, depending on the severity of the initial symptoms, for the pain to disappear after pure air is reached.

The degree to which the different symptoms develop differs even in severe cases. This may be due to differences in the method of breathing, for the serious involvement of the nasal passages is less likely to occur in the case of mouth breathers. The pain in the eyes is somewhat different from the smarting caused by the simple lachrymators, the impression being gained that small gritty particles have been driven into the eyes.

**78.** A very early condition sometimes complained of is giddiness, and in a certain number of cases consciousness is quickly lost and a comatose condition persists for several hours; others, without losing consciousness, pass into a lethargic condition for a period of from 12 to 24 hours. A remarkable feature in these severe cases is the intense mental distress which accompanies the symptoms already described. Even slight cases feel and look miserable until the irritation passes off, and the picture of utter dejection and hopeless misery furnished by severe cases has no counterpart in any

other type of gas poisoning. Occasionally the physical depression results in the temporary loss of mental control, and men have been known to act as though driven mad by their pain and misery.

79. In addition to the above changes in mental condition, alterations in motor power occur sometimes within an hour but in some cases are delayed in appearance. The commonest complaint is of formication mainly referred to the finger tips—and this has been observed in laboratory workers who would not be likely to exhibit functional phenomena—but temporary paralysis involving one or more limbs is seen in a fair number of cases. This paralysis rapidly disappears and in 24 hours there is no sign of it left. These cases are a strong argument in favour of a central toxic action of the gas affecting the cortical or spinal centres leading to a temporary abolition of function. In other cases a more generalized motor weakness appears.

80. These motor changes in the early stages must be clearly differentiated from the sensory changes, which occur later in the course of the illness. The former may, with some degree of certainty be ascribed as toxæmia, transitory but definite, of the central nervous system. It is far more difficult to come to a certain conclusion about the latter. The sensory changes are mainly a disturbance of sensation leading to anæsthesia more or less complete, a condition which commonly supervenes about the fourth day. The extent of anæsthesia varies from a mere numbness of the finger tips to a complete loss of sensation over a considerable part of one or more limbs. Commonly the affection is bilateral and conforms to the glove and stocking distribution. In these cases the reflexes are unaffected and the sensory condition is not accompanied by any motor change or any sign of trophic disturbance. Pressure in the nerve tracts is not painful nor is any evidence of peripheral neuritis obtainable. Naturally, the first suggestion is that an arsenical neuritis is present, but prolonged and careful observations have tended to disprove this view. Progress in all these cases is towards recovery, and in no instance has any indication of involvement of motor nerves developed. Numerically, cases showing this manifestation are very uncommon.

81. The probability is that these sensory changes are functional in character. The distribution of the anæsthesia is never of a segmental character but is almost always of that glove and stocking distribution which is so significant of functional disorders. Also, recovery is so rapid and so uniform



that this forms part of the argument against an organic basis for the lesion. Whilst a final conclusion as to the true nature of these nervous phenomena is difficult to arrive at, since the dividing line between functional and organic changes is one which it is impossible to demarcate with firmness and accuracy, it is certainly true that no lasting organic lesion is produced.

In some cases there are symptoms in the respiratory tract in the shape of a mild and evanescent bronchitis. A rise of temperature is extremely uncommon.

The above description of the symptoms may be taken as applying to the most severe type of case met with in the field, but such cases are in the minority, and in many instances the irritant effects are so slight and transitory that the affected men never leave their units. The progress of patients affected by the arsenical warfare gases, from whatever source, is in the main uniformly good and rapid to convalescence. The symptoms abate within 24 hours, and by 48 hours the great majority of cases have practically recovered. The last symptoms to disappear are gastric discomfort and pain in the nose and forehead, and when these have cleared up, beyond a general weakness with no physical signs and a certain amount of mental depression or mental irritability, the whole condition disappears within a week. So rapid is the progress on the whole that it was part of the army routine in France to retain the cases in the forward area rather than evacuate them to the base, and the great majority were fit for duty within ten days.

**82. Treatment.**—The respirator, properly worn, will protect from sensory irritant gases.

In the early stages pain is the one symptom calling for relief, beyond the general principles of treatment of all gas cases—rest, fresh air, removal of contaminated clothing and equipment, and light diet, preferably a fluid one. The pain is best relieved by inhalations of chloroform which may be put up in dark glass ampoules holding 1 or 2 c.c.

For the pharyngeal irritation and pain, glycerine or menthol jujubes were found useful.

In the later stages treatment should be directed to toning up the physical and nervous systems.

It must not be forgotten that quite different symptoms of true arsenic poisoning may arise from the drinking of water, contaminated with the arsines, from shell holes. The use of shell-hole water for drinking or washing purposes should be prohibited.

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## CHAPTER V

### LACHRYMATORS

83. The lachrymators are a group of chemical warfare agents which, in low concentrations, produce watering of the eyes in a moment or so, and in rather greater concentrations produce pain and smarting of the eyes with spasm of the lids so that it is impossible to keep the eyelids open. The consequent interference with vision is quite sufficient to put a man temporarily out of action. As the concentration of the vapour increases, further effects show themselves. The irritant action on the respiratory passages and lungs is rendered evident by a burning sensation in the throat, and burning pain or discomfort in the chest, and unless the affected men can withdraw from the poisonous atmosphere, nausea, retching and vomiting soon commence, accompanied by pain in the epigastrium. A man affected to this degree may appear dazed and become somewhat torpid, mainly, perhaps, from the general intense discomfort and the repeated retching and vomiting, though a direct effect of absorbed vapour may also play a part.

84. Speaking generally, the casualties that resulted from the German lachrymator shell in France never exhibited more severe symptoms than the above, though a few instances occurred when death was caused by the bursting of one of these shells close to a man, and there was uncertainty, in the absence of post-mortem examination, how far the fumes were a contributory cause of death in addition to actual violence. Many of the troops experienced simple lachrymation, but the actual number of casualties that reached medical units was small, owing to the limited number of lachrymator shell used, and the high boiling point of the liquid prevented any high concentration being attained in the open air. The striking feature was the rapidity with which the cases recovered when once they had withdrawn from the poisonous atmosphere. The symptoms of those who were more severely affected had usually abated by the time they reached the advanced dressing-stations ; at the end of 12 hours there was little wrong with them, and even the redness and swelling of the eyelids and injection of the conjunctivæ, which had been conspicuous in the early stages, had nearly disappeared.

In one instance, when death occurred within 24 hours after exposure to the fumes as the result of accidental explosion of a shell, autopsy revealed œdema and congestion of the lungs, and acute inflammation of the whole of the air passages from



the larynx downwards, which increased in intensity in the smaller bronchial tubes, the surface of the mucous membrane being covered and the smaller tubes blocked by a viscid, purulent exudation.

85. Occasionally, workers in factories engaged in the handling of ethyl iodoacetate have suffered severely from the poisoning, and fatal cases have occurred. Exposure to concentrated fumes causes severe conjunctivitis, and if inhaled, inflammation of the respiratory passages may result. The fumes will also cause blistering of the skin and abrasions exposed to them very frequently become septic.

86. There are several compounds which may be used as lachrymators, both liquids and solids. Ethyl iodoacetate is an example of a liquid, and is classified from the tactical point of view as a semi-persistent substance. This substance is easily dispersed in bombs and shell. Chloroacetophenone is an example of a solid and is a powerful lachrymator in high concentrations. In addition to the eye effects it causes a burning and tingling sensation of the skin. It can be dispersed by generators, in a similar way to the arsenical compounds, and when dispersed in this manner is classified as a non-persistent lachrymator.

87. **Treatment.**—The respirator, if properly applied, affords perfect protection.

No treatment, other than simple lavage of the eyes, was found necessary.

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## CHAPTER VI

### DIRECT POISONS OF THE NERVOUS SYSTEM

#### Hydrocyanic Acid Gas

88. The most important of these is hydrocyanic (prussic) acid gas. The actual concentration of the gas is of far greater importance than the duration of exposure. Professor Barcroft has proved by personal experiment that a concentration of 1 part of gas in 2,000 would have to be attained before much effect would be produced on troops in the field. Concentrations of the gas below 1 part in 10,000 have little or no effect.

When the quantity of gas in the air is small, it may be borne

for a considerable time without ill-effects, but once the poisonous concentration is reached, death is very rapid. Unless the dose is a fatal one, recovery from the toxic effects is rapid, and no serious after-effects need be feared.

**89. Mode of action.**—When death occurs it is attributable mainly to direct paralysing effect on the respiratory centre and the heart. The substance temporarily stops all oxidative processes in every cell of the body to which it has access, but the tissues may recover their full vitality; if the circulation and respiration can be maintained, the poisonous substance is quickly eliminated. According to French observers there is marked constriction of the bronchial tubes by spasm of the muscle in cases where rapid death takes place. Apart from this there is no obvious pathological change. When death is caused by inhalation of this gas it is unlikely that the smell of the gas will be detected at the autopsy, as may be the case when poisoning is due to the ingestion by the mouth of a large dose of prussic acid.

**90. Symptoms.**—If the concentration of the gas is much below that which causes rapid death, only giddiness may be produced. If, however, the concentration is high, the symptoms follow in rapid sequence: giddiness, confusion, headache, failure of vision, palpitation, and pain in the chest and over the heart culminate in a few seconds in loss of consciousness, the respirations become laboured, being slow with long-drawn-out inspirations, convulsions set in and death ensues in one or two minutes from failure of respiration and then of the heart.

Still higher concentrations cause immediate unconsciousness, dilatation of the pupils, a few gasping respirations and death with or without convulsions.

**91. Treatment.**—The respirator, if properly adjusted, affords complete protection.

The treatment must be immediate if it is to be effective. The case must be dragged at once into the fresh air, and if the respiration has stopped or if it is weak and gasping, artificial respiration must be commenced, coupled with stimulation by splashing cold water on the face and chest, and by friction of the limbs.

This same treatment must be carried out for any casualty produced by a gas of this group.

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## CHAPTER VII

## POISONOUS GASES NOT USED FOR OFFENSIVE PURPOSES, BUT LIABLE TO BE ENCOUNTERED BY TROOPS

## Carbon Monoxide

92. **General.**—This gas is formed in large volumes on the explosion or detonation of explosives, also when the combustion of carbonaceous material takes place in a limited supply of air. It may be met with under the following circumstances :—

- (a) Mining operations.—The galleries may be flooded with the gas after the explosion of one of our own or of an enemy's mine or camouflet, or as the result of the explosion or burning of a blasting charge. The gas is liable to be driven into the ground disturbed by the explosion and to come welling out later, especially if the barometer commences to fall after the explosion, or it may be met with in the form of pockets of gas when new galleries are driven through ground disturbed by a previous explosion. This gas has even been known to be driven out of the shafts into adjacent trenches after the explosion of a mine.
- (b) Fumes from high explosive shell.—When a high explosive shell penetrates the soil and bursts in the neighbourhood of a deep dug-out, or other relatively confined space, the carbon monoxide generated in the explosion may find its way into the dug-out and poison the occupants.
- (c) Fumes from the firing of guns.—Cases of carbon monoxide poisoning sometimes occur in closed machine-gun emplacements or in gun pits owing to the blow-back from the gun.
- (d) Burning buildings, &c.—There is considerable risk of carbon monoxide poisoning in the interior of burning buildings. Very serious amounts of this gas may be formed if the timbering of deep dug-outs or mine galleries catches fire.
- (e) A burning coke brazier gives rise to carbon monoxide and is a great source of danger in a small ill-ventilated billet or dug-out.
- (f) Carbon monoxide may be found in the interior of tanks, especially in the vicinity of the exhaust

side of the engine, and is due to the escape of exhaust gases from the engine and to blow-backs from the gun, or in the interior of ambulance or closed motor cars if the exhaust gases leak into the vehicle.

- (g) The exhaust gases of motor engines contain this gas, and such engines are dangerous if installed in ill-ventilated dug-outs or cellars.

93. The great danger from carbon monoxide lies in the fact that it is colourless, odourless and non-irritant, and that the onset of symptoms is so insidious that very often the first warning that a man may receive is failure in the power of the limbs, which will prevent him from retreating into safety.

94. **Mode of action.**—This gas owes its poisonous properties to the fact that it combines with hæmoglobin to form a dissociable compound just as oxygen does, though the affinity of carbon monoxide for hæmoglobin is 240 times that of oxygen.

When a man breathes air containing a small proportion of carbon monoxide, a partition of the hæmoglobin of the blood between the two gases occurs, the final partition being determined by the relative concentration of the two gases. As the saturation of the hæmoglobin with carbon monoxide increases, its oxygen carrying power progressively diminishes and symptoms of anoxæmia begin to show themselves. There are no changes in the lungs hindering the access of oxygen to the blood as there are in the case of poisoning by an acute lung irritant gas.

95. **Morbid anatomy.**—At the autopsy the blood may be red in colour instead of dark if there is a considerable degree of saturation of the hæmoglobin with carbon monoxide. If the case has continued to breathe for some time after reaching an atmosphere free from carbon monoxide, this gas will have been partly or entirely displaced from the hæmoglobin and the blood after death will have its normal colour.

The simplest method of detecting the presence of carbon monoxide in blood is to compare the colour of a diluted solution of the suspected blood with a similar solution of normal blood. Take a drop or two of the blood from the finger of a normal person and dilute it in a test tube very considerably with water (a  $\frac{1}{2}$  per cent solution is a convenient strength), so that when examined by transmitted daylight the colour of this solution is a reddish yellow. Then take a drop or two of the suspected blood and dilute it similarly with water so that the *depth* of colour of the solution is the same as that of the solution of normal blood when both are viewed by



transmitted light. On examining the quality of the colour it will be found that the solution made with the suspected blood, if it contains carbon monoxide hæmoglobin, is definitely pinker than that made with the normal blood, though it will not have the full pink tint of the same normal blood solution if the latter be shaken with coal gas so as to saturate it completely with carbon monoxide.

The lungs show no abnormal changes in cases of rapid death. Small punctate hæmorrhages may be found in the white matter of the brain, and sometimes ecchymoses in the meninges and even large cerebral hæmorrhages if the case has been exposed to a concentration of carbon monoxide sufficient to cause prolonged unconsciousness.

**96. Symptoms.**—Except with very massive doses, when loss of consciousness is very rapid, the symptoms develop gradually as the gas is only absorbed slowly. In a concentration of one part in a thousand, if a man is at rest it will take two hours before giddiness appears, and he will not be definitely disabled until two and a half hours have elapsed. The rate of absorption of the gas is much accelerated by muscular exercise owing to the increase in the breathing and the circulation rate, while the symptoms become accentuated since the oxygen requirements of the body are much greater than at rest. With a concentration of two parts in a thousand a man will be seriously affected in half an hour if he is performing a moderate amount of muscular work, and this concentration may prove fatal with prolonged exposure.

**97.** Small animals, *e.g.* mice or canaries, are far more rapidly affected by carbon monoxide than is man, owing to their relatively high rate of metabolism, which is 15 times that of man. If such animals are used to give an index of the presence of carbon monoxide in a suspected atmosphere, it must be remembered that though they show symptoms long before a man feels any effects, the man will in the end be reduced to the same condition as the animal, and he ought, therefore, to leave the dangerous atmosphere directly the animal shows signs of being affected, unless he is protected by a special apparatus.

**98.** With moderate doses, the symptoms, which are essentially those of anoxæmia, develop in the following sequence. The first sign frequently is a feeling of loss of power in the limbs. Giddiness, slight confusion of mind, and breathlessness and palpitation on the least exertion show themselves. The confusion of mind and loss of power in the legs often preclude a man from withdrawing from danger even though he is dimly aware that safety is only a few yards

distant. The failure of power in the limbs and the mental confusion rapidly increase and the man may appear drunk, shouting incoherently, laughing, swearing and praying. Apathy and complete helplessness supervene, and failure of the intellectual powers gradually passes into complete unconsciousness which may finally terminate in a painless death.

In a mild case of gassing with carbon monoxide a severe headache, accompanied by nausea, is very likely to develop.

**99. Treatment.**—It must be borne in mind that the box respirator does not afford protection against carbon monoxide; protection can only be attained by the use of special oxygen breathing apparatus (Proto or Salvus sets).

The case must be carried at once into the open air. Any increase in the oxygen requirements of the body must be avoided, and the case therefore kept at rest. The other essential is the proper administration of pure oxygen, for from  $\frac{1}{2}$  an hour to 1 hour, care being taken that the expired air is not rebreathed. This can be carried out with a Novita oxygen apparatus such as is kept at mine rescue stations, or by means of the nitrous oxide anæsthesia apparatus; as an alternative the Haldane oxygen administration apparatus may be used with the oxygen delivery set at 8–10 litres a minute.

Very good results have also been obtained by breathing air enriched with carbon dioxide to the extent of 5 to 7 per cent, the rationale of this treatment being that the high percentage of carbon dioxide stimulates the respiratory centre causing increase in the depth and rate of breathing and thus helping the elimination of carbon monoxide.

If the breathing is very shallow, administration of oxygen may be combined with artificial respiration. The case should not be evacuated until this has been carried out.

Collapse should be combated by external warmth and by friction of the limbs.

**100.** Cases of carbon monoxide poisoning have been known to recover, even when they have remained unconscious for so long as 48 hours after removal from the poisonous atmosphere. In cases that have been severely gassed, the possibility of subsequent cardiac dilatation must not be lost sight of, and cases of severe gassing should not be returned to duty until confidence is felt that the circulation has recovered from the strain. As a result of damage to the nervous system while the blood was charged with carbon monoxide, paralysis of single muscles, or groups of muscles, or different forms of mental disturbance, are sometimes found as sequelæ.



## DEFICIENCY OF OXYGEN

101. **Danger arising from a simple reduction of the Oxygen percentage in the Air.**—It should always be remembered that the air in wells, unventilated or disused mine galleries, underground shafts or chambers may be seriously deficient in oxygen, and that it may therefore be extremely dangerous to enter such places rashly. The reduction in oxygen percentage is caused by the oxidation of organic or mineral matter in the soil, and it may or may not be accompanied by the accumulation of carbon dioxide according to the precise nature of the oxidation.

102. If the deficiency of oxygen is moderate little abnormality may be experienced save, perhaps, for a feeling of dizziness or weakness of the limbs whilst doing muscular work, symptoms which may pass off rapidly if the work is stopped for a few minutes. If, in addition to the deficiency of oxygen, excess of carbon dioxide is present in the air, attention will be called to the fact by the unusual panting which will result. Not infrequently, however, the deficiency of oxygen may be so great that entry into such an atmosphere will result in immediate asphyxia with no warning symptoms. When the barometer happens to be rising the air in one of these places may be perfectly safe to breathe, and yet an hour or two later, if the barometer begins to fall, the oxygen may be grossly deficient, for, with a falling barometer, air, from which the oxygen has been largely abstracted, tends to well out of the soil into the adjacent cavity.

103. The essential thing to bear in mind is to test the air in any place about which doubt is felt before allowing a man to enter it. If a lighted candle is lowered into the place to be tested and remains alight the air will be safe to breathe; if it is extinguished steps must be taken to ventilate the place before attempting to enter it, or else some self-contained breathing apparatus (*e.g.* a mine rescue apparatus) must be worn by those who descend the shaft or enter the gallery. In any case if there is the slightest suspicion about the quality of the air—though good at one part of a gallery it may be bad at another—any one who has to enter for testing purposes should have a life-line attached to him so that he may be pulled out of danger at once in the event of an accident. Naturally, the lighted candle test must not be used in old coal workings, where fire damp (methane) is probably present, for fear of causing an explosion.

104. There is also a serious risk of gross reduction of oxygen

in the atmosphere in closed compartments, ships' holds, &c., in which grain or other oxidizable substances, *e.g.* coal or vegetables, are stored, owing to the absorption of oxygen, unless adequate ventilation is provided. It should not be forgotten in this connection that freshly painted surfaces and even rusting iron may absorb a large quantity of oxygen.

105. The only remedy if a man falls unconscious in an atmosphere deficient in oxygen is to pull him out into fresh air at once, and if the breathing has stopped to apply artificial respiration. Would-be rescuers must, however, themselves remember the danger of the atmosphere: it is no use adding to the toll of casualties uselessly, and it is for this reason that the provision of a life-line is recommended.

### **Nitrous Fumes**

106. Though these fumes caused no material cases of poisoning in the warfare on land, a number of instances were reported in the Navy owing to the liberation of these fumes from burning cordite.

The mode of action of this gas is described in Chapter X (para. 142).

107. The treatment is that described in Chapter III for lung irritant gases.

### **Arseniuretted Hydrogen**

108. This gas has been met with in submarines during the war when casualties occurred. Its origin was traced to the use of impure material in the battery. A detailed account of its mode of action and symptoms will be found at para. 146 *et seq.* of this manual.

The service respirator protects against arsine for short periods.

### **Phosphorus Burns**

109. White phosphorus is used in the field primarily to produce smoke for screening purposes. When released from shell solid particles of phosphorus are widely scattered. These melt and catch fire, flaming vigorously when exposed to the air, and will cause severe burns if they land on personnel. If the phosphorus has landed on the clothing, the clothing should be immediately ripped off. The principle upon which the first-aid treatment depends is to exclude the flaming mass from the air. This is most readily and practically done by smothering the mass with anything that is available. The best thing ordinarily available is earth, but a rag may be effective.



If water is available the part should be totally immersed. Small quantities of water applied cause the phosphorus to splutter. A compress of 2 to 5 per cent copper sulphate will cause the phosphorus to cease flaming by forming a coating of copper on the surface of the mass.

Phosphorus melts at  $112^{\circ}$  F., so that if the part is immersed in warm water the molten substance can be removed under water, or wiped off with a gauze sponge held in a forceps. Great care should be taken to remove every particle.

Subsequent treatment follows the lines adopted for ordinary thermal burns, but oils or fatty dressings should not be applied unless it is certain that all the phosphorus has been removed.

**110. Poisoning by Aromatic Nitro Compounds used in the Manufacture of High Explosives.**—Although a consideration of these subjects does not come directly under the heading of Chemical Warfare, medical officers may from time to time be called upon to treat patients suffering from the effects of intoxication by them.

The toxic effects of the nitro-benzenes and nitro-toluenes are very similar, but the greater the number of nitro-groups in the molecule the higher is the toxicity. Further, the chloro-nitro derivatives are more dangerous than the simple nitro compounds.

The condition produced may vary from a general malaise with loss of appetite, headache, giddiness, and constipation, to dermatitis or severe gastritis, toxic jaundice, and death.

**111. (T.N.T.) Tri-Nitro-Toluene.**—The effects of poisoning by T.N.T. may be taken as typical of the more severe affections.

The poison is absorbed not merely by inhalation but much more through the skin. The ill-effects produced by this substance may be classified as follows :—

- (a) Dermatitis.
- (b) An early, probably reflex, vomiting.
- (c) Affections of the blood or blood-forming organs.
- (d) Toxic gastritis.
- (e) Toxic jaundice.

**112. Dermatitis.**—Workers vary greatly in their susceptibility to this condition. The parts most frequently affected are the wrists, ankles, and neck where rubbing with contaminated clothing occurs; but the facial and intercrural regions may also suffer.

The rash is usually a superficial follicular or widespread erythema, but on the hands a chronic eczema or cheiropompholyx type of eruption is common.

Secondary infection and thrombo-phlebitis are sometimes seen, though as a rule the skin affection yields readily to simple treatment. In all forms itching is often intolerable. Yellow staining of the hands and occasionally of the feet of T.N.T. workers is very common.

In very few cases do the same patients suffer both from dermatitis and constitutional ill-effects. Cuts and abrasions of the skin heal slowly in workers exposed to T.N.T.

113. *Functional or reflex Gastritis*.—Among new workers, vomiting in the early morning and occasionally after food is a common complaint during the early days of employment. This condition, which is probably psychic in origin, rapidly passes off.

114. *Toxic Gastritis* is the outstanding ill-effect in factory workers with this substance. Pain is the most general and often the only complaint. This pain is always local in the epigastrium or lower chest, and varies from discomfort to pain of great severity. It is colicky in character and has no relation to meals. Rest relieves it, vomiting rarely coincides with the pain and does not give relief, nausea and aversion from food are concomitant symptoms; constipation is the rule in these cases.

On examination these patients are apathetic and show muscular weakness; their faces are pallid, drawn and wizened; their sclerotics are dulled, their tongues are clean. High-coloured urine and frequency of micturition are common. Cyanosis of the lips and tongue are generally noticeable. There is some epigastric tenderness. These symptoms may come on at any time from a fortnight to nine months after employment on T.N.T. Two to six weeks may elapse before the patient is again fit for work.

Open air and purgatives are the best treatment. Relapses are uncommon, but if they occur are an indication that the patient should not be allowed to carry on with this type of work. Fear of return to work on shell-filling may lead to chronic invalidism.

115. *Toxic Jaundice*.—Young adults are frequently attacked, and when attacked experience a high mortality, so that employment on T.N.T. of workers below the age of 18 should be prohibited. The onset of jaundice may occur immediately after commencing employment, or may be delayed for months.

The early signs and symptoms of this condition are of paramount importance, since the prognosis depends to a great extent on early removal from work.



Early symptoms are dizziness, tiredness, headache and an overpowering desire to sleep in the daytime. Dyspepsia, abdominal pain and vomiting are sometimes present. On the other hand the fatal toxæmia may develop without warning.

In many cases an absolute diagnosis of hepatitis is difficult in the early stages, but valuable evidence may be gained by a simple laboratory test. A little blood of the suspected worker is withdrawn into a collecting tube and allowed to coagulate. The serum above the clot shows occasionally a vivid yellow colour. A worker whose blood gives such a reaction should be removed from contact with T.N.T. without delay.

Vomiting in cases with toxic jaundice is often severe, recurring with each attempt to take solid food. Constipation with white hard stools is frequent. Abdominal pain when present is generally in the liver region; and marked tenderness may be elicited by pressure in the liver area. The area of liver dullness may be diminished.

116. *The Blood Changes.*—The commonest result is an alteration in the hæmoglobin (which is perhaps changed to methæmoglobin), so that the patient's lips and ears assume a cyanotic tint. With this there is no change in the blood count.

Much more rarely a grave anæmia of an aplastic type may develop. This has no direct relationship to the toxic jaundice.

The disease may show itself as a gradually increasing debility and breathlessness as found in pernicious anæmia, or the onset may be sudden and hæmorrhagic. The hæmorrhage may show itself first in the legs as a general purpura, or there may be a sudden bleeding, *e.g.* in the case of women workers, from the uterus.

117. *Preventive measures.*—These consist in the strictest observance of regulations framed to reduce or eliminate contact between the workers and the toxic material. Workers should wear overalls fitting closely at the neck, wrists and ankles, leather gloves, caps, and shoes fitted with wooden soles. It is specially necessary that the inside of the gloves and overalls should be inspected to ensure that they have not been contaminated by the toxic powder. When necessary, respirators should be worn.

Bathing should be compulsory before the workers put on their ordinary clothes to leave the factory.

No food should be taken in the workrooms, but canteens and mess-rooms should be provided. Before going to these, workers should wash thoroughly and change their clothes.

A most important measure is the provision of a thorough ventilation system, the best practice being when the extraction of air is arranged at floor level.

Filling devices, which obviate handling the toxic materials, should be developed.

118. Medical inspection of all workers should be frequent and thorough. In suspicious cases a blood count for anæmia and the Van den Berg test of the blood for bile pigment should be carried out.

*Remedial measures.*—In mild cases a few days' rest, and the free use of aperients are usually all that is necessary. If considered desirable, a change of employment should be made. As soon as there is any indication of the onset of toxic jaundice, the patient should be removed from contact with the toxic material without delay, and given absolute rest in bed. Frequent doses of Mist. Alba or a similar aperient should be given, and the diet should be of the lightest, or simply of milk.

## CHAPTER VIII

### MISCELLANEOUS

119. The most important feature in safeguarding the individual against effects of chemical weapons is the protection of the eyes, respiratory passages and lungs. The service respirator will give complete protection from all chemical warfare gases likely to be met with in the field. It should be remembered, however, that the respirator gives no protection against carbon monoxide (*see* Chapter VI). A description of the service respirator is not given in this chapter but may be found in Defence against Gas; only the purely medical aspects are discussed in the succeeding paragraphs.

120. In addition to the effects which gases have on the eyes, respiratory passages and lungs, gases belonging to the vesicant group also affect the skin. Ordinary clothing being porous is unable to keep out vapours or liquids, and protection must be sought either by impregnating fabric with some neutralizing substance or by providing non-porous clothing. Hitherto impregnating clothing has not proved a practical proposition. The material which has proved most effective is a closely woven cotton fabric treated with a drying oil, such as linseed which renders it non-porous. Clothing made of



such material is known as Protective Clothing. For a further description see Defence against Gas.

The use of clothing of this nature is limited because the free loss of heat from the body by evaporation of sweat is interfered with owing to the lack of ventilation, and also because it hampers movement to a considerable extent. A general issue cannot, therefore, be made. It is, however, of the greatest use for selected individuals employed on duties which render them especially liable to gross contamination, for example, personnel engaged on the work of decontamination, and orderlies removing contaminated clothing from patients.

**121. The Physiology of the Respirator.**—In determining the effects of the respirator on the wearer three main factors have to be considered :—

- (a) The psychological effect.
- (b) The effect of dead space on breathing.
- (c) The effect of resistance to breathing.

*The Psychological Effect.*—When an individual first puts on a respirator he feels a certain amount of restriction of his breathing. These feelings are to a great extent subjective, being suggested by the fact that the respiratory passages are covered and that, unconsciously perhaps, the individual expects his supply of air to be restricted because he is unaccustomed to breathing against a resistance. They are also, no doubt, to a slight extent due to the nervous stimulus to respiration given by the slight resistance which the respirator offers to breathing, necessitating an increase of effort on the part of the respiratory muscles.

As the wearer becomes accustomed to the apparatus, however, his breathing returns to the normal and he experiences no difficulty in obtaining all the air he requires, even during heavy work.

Much depends upon judicious training from the first, and the instructor should explain that the difficulty in breathing is only due to inexperience.

*Dead Space.*—In designing the respirator consideration has been given in the case of the Tissot type of facepiece to reducing the dead space between the mask and the face to a minimum, so that the wearer is not called upon to rebreathe his own expired air to any significant extent.

*The Effects of Resistance.*—As has been mentioned above, even a slight resistance to breathing calls for increased work on the part of the respiratory muscles, and so puts a slight strain upon the individual. This additional strain is shown by the increased response of the pulse rate to exercise when wearing

a respirator, and delay in the time taken to return to the resting rate after completion of the work. Such an increase is shown even by a well-trained individual who is thoroughly accustomed to wearing a respirator, and it is, therefore, to be expected that the strain will be greater in the untrained or tired individual and in the individual unaccustomed to its use.

The initial discomfort in breathing experienced when the respirator is first put on quickly disappears, and prolonged training enables the individual to work without ill-effects with a respirator up to a resistance of 4 inches of water at a flow of air of 3 cubic feet a minute through the container, though, of course, the lower the resistance the greater his comfort.

**122. Hygiene of the Respirator.**—The respirator is an article of personal equipment, the facepiece remaining in the possession of the individual throughout his services and the container being changed as often as is necessary. Facepieces returned to store may be re-issued.

To ensure that these appliances are maintained in a hygienic condition, regulations lay down that they are to be disinfected half-yearly and, also, when exchanged, and cleansed after each wearing. It is also of importance that medical officers in charge of effective troops should take steps to ensure that disinfection is carried out when a man is admitted to hospital suffering from any disease where infection is likely to be carried by the respirator.

The details of the procedure to be carried out in disinfecting respirators is given in Equipment Regulations, Part I, 1923, para. 304. Briefly the procedure is as follows:—a solution of a disinfectant approved for the purpose is made up freshly to the required strength. The rubber connecting tube is detached from the container, and the whole facepiece with connecting tube is immersed in the disinfecting solution. Any convenient vessel may be used to accommodate a number of facepieces at one time. After five minutes the facepiece is removed from the disinfecting solution and thoroughly washed out with water. As much water as possible is shaken off the facepiece and out of the connecting tube. The whole facepiece is then laid out to dry. The time taken to dry will vary with the time of year, but it is essential that all moisture is dried out of the connecting tube before replacement on the container.

For routine cleansing the container should not be detached from the facepiece, but care should be taken to prevent water, etc., entering it. The whole of the inside of the facepiece should be swabbed over with a rag moistened with a disin-



fectant solution approved for the purpose. The facepiece should then be washed out with water and allowed to dry.

In the case of men working under high temperature conditions, inconvenience may be experienced by the pressure of the rubber on the moist skin of the forehead. Under these special conditions, the use of the following astringent antiseptic powder may be found useful :—

Zinc oxide	..	..	..	40 per cent.
Boracic acid	..	..	..	10 per cent.
Starch	..	..	..	50 per cent.

It is essential that this powder be dusted on the skin of the forehead only and not applied inside the facepiece.

**123. The Protection of Helpless Patients.**—It is the duty of the medical officer or orderly to see that the protective appliances of his patients are in good order and in readiness for use ; and it must be remembered that in future this may apply not only to forward areas but also to areas on the lines of communication or at the base which may be subjected to gas bombardments from the air. It is particularly necessary to ensure the safety of patients who cannot help themselves. In this connection it is to be remembered that an unconscious person can breathe comfortably in the present facepiece.

**124. Dressing of Head Wounds when the Respirator is worn.**—Where the respirator has to be worn in the case of patients suffering from head wounds, adjustment of the respirator to ensure gas tightness is of first importance.

Gas tightness of the respirator can be obtained by ensuring contact between the fitting surface of the facepiece and the skin which lies over the bony structure of the forehead, cheeks and chin. Any dressing applied between the fitting surface and the skin will permit the entrance of gas and, therefore, no dressing should be placed over these parts if this can be possibly avoided. Should it be found absolutely necessary to place a dressing on a wound over these parts, the minimum amount of gauze should be used. It may be found necessary to remove some of the padding over the wound in order to ensure sufficiently close fitting of the facepiece. It will be found that by correctly adjusting the harness tension by first loosening off the elastics, applying the dressing and then gradually tightening up, that the harness itself will act as an excellent substitute for a bandage.

**125. The Physiology of Protective Clothing.**—The chief limiting factor in the use of protective clothing is the fact that the normal heat loss from the surface of the body is interfered with.

Body temperature is regulated by the balance between heat loss and heat production. Heat loss occurs chiefly through radiation and conduction from the surface of the body and by evaporation of moisture from skin and lungs.

When the body is clad in impervious garments, free circulation of air is prevented and loss of heat through convection is checked. Air itself is a bad conductor, and between the body and the suit there is a layer of still air. This confined air soon becomes saturated with moisture, and heat loss by evaporation then ceases. If the condition persists for long the body temperature rises, the heart beat becomes rapid and the individual experiences discomfort and distress even up to complete prostration and collapse.

Climatic conditions, such as temperature and relative humidity, have a distinct bearing on the length of time the suit can be worn with comfort. In warm atmospheres cooling of the body may be assisted by douching the suit with cold water.

No clothing other than underclothing should be worn under the suit.

If it is necessary that the individual should wear the hood, it will be found that symptoms of heat exhaustion occur very much earlier, and the working spell will be greatly reduced compared with that of a man doing similar work under similar conditions, but with the neck and that portion of the face not covered by mask left free.

Men trained to work in a respirator and protective suit can do long spells of manual labour, varying from half an hour to four hours, depending upon the atmospheric conditions and the nature of the work. But a man equipped also with the special hood may be unable to work longer than fifteen minutes in a hot atmosphere.

**126. Protection of Food and Water Supplies.**—Where food unprotected by tin containers has been exposed to contamination by liquid mustard, lewisite or the arsenical warfare gases, it should be destroyed. Tinned foods are safe to use after the tins have been decontaminated. The gaseous vapours alone will not render food harmful, but high concentrations will in some cases render such food unpalatable.

Water contaminated with arsenical preparations is dangerous for washing, cooking and drinking purposes.

Water contaminated with mustard gas.—When liquid mustard gas is mixed with a large quantity of water, some of it dissolves, and is rapidly hydrolysed to form harmless compounds. The amount dissolving is usually small and will depend upon the amount of agitation and the temperature.



The bulk of the liquid soon falls to the bottom and will remain there as oily globules for long periods.

Reservoirs are in the majority of cases uncovered, and there will always be the possibility that mustard spray may alight on the water, or that a mustard bomb may be dropped into it.

As regards the first eventuality, experiments indicate that risk of dangerous concentrations of mustard gas reaching the domestic water supply would in all probability be remote, for the dissolved mustard gas would rapidly be rendered harmless by hydrolysis, and the undissolved material, so long as the water was not violently disturbed, would lie at the bottom practically inert except for very gradual solution. If the undissolved mustard gas were carried by any means on to the sand filter, it would be retained there, as liquid mustard gas does not pass readily through wet sand.

It seems likely that the effects from bursting in water a bomb which contains mustard gas would be similar. For if the mustard gas were dispersed in very fine particles, it would tend to be hydrolysed rapidly, whilst coarse particles would tend to sink and remain relatively inert.

Though danger from this source would appear remote, it is possible that extensive contamination might taint the water as regards its taste, for when water is experimentally contaminated with dissolved mustard gas and then boiled or allowed to stand for a time, so that it becomes harmless owing to hydrolysis, it still possesses a disagreeable taste.

The above considerations apply only to stored and filtered water, such as that from a reservoir. A running stream might be dangerous for a considerable distance below the point of contamination, for droplets of mustard gas might be carried with the stream. Further, water from shell holes or shallow ponds likely to have been contaminated by mustard gas should not be used for drinking or washing purposes.

Whatever the character of the water supply, it would be an advisable precaution to issue instructions that all water for household purposes should be boiled before used, in any case of suspected contamination by mustard gas.

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## CHAPTER IX

PREVENTION AND HANDLING OF CASUALTIES  
FROM CHEMICAL WEAPONS

127. **General.**—In chemical warfare the element of surprise and the effects which gas has on morale are factors of great importance. Surprise can be combated by prolonged training in defensive measures, particularly as regards wearing respirators and by strict discipline.

The effect on morale is more difficult to deal with, since it is brought about both by the fear of the unknown and also by the strain which constant watchfulness for gas alarms imposes upon troops.

128. The best measures to adopt in maintaining morale are :—

- (a) Careful and thorough teaching upon the uses and limitations of chemical weapons, and
- (b) Watchfulness on the part of medical and other officers for the incipient signs of nervous strain. As soon as these appear in any individual he should be given a rest or removed to a locality of comparative safety.

Moreover, medical officers should remember that all men reporting themselves as gassed are not necessarily genuine cases. Men may quite honestly believe themselves to be gassed when in reality they have only smelt the fumes of high explosive, or of more toxic substances in such concentrations and for such a length of time as will do them no harm. Such cases require firm handling and no patient should be labelled as a gas case without careful examination and sifting of evidence.

129. Casualties from chemical weapons may be classified as follows :—

- (a) Slight cases.
- (b) Early acute cases.
- (c) Late acute cases.

In each class correct treatment and disposal depends upon accurate knowledge on the part of medical officers, both as regards the clinical conditions to which poisoning gives rise, and also as regards the tactical uses of this arm.

The slight cases include those mentioned above, cases suffering from the effects of lachrymators and a large percentage of the cases of exposure to toxic smokes, *e.g.* the arsenical compounds.



For many of these, removal from the toxic atmosphere, rest and refreshment are all that is necessary, and the majority do not require evacuation beyond the divisional area.

Exposure to arsenical compounds may, however, in some cases result in acute illness which renders the patient unfit for duty for a period varying from a few days to a fortnight.

The early acute cases are those arising from exposure to the lung irritants, and the necessity for care in the time and mode of evacuation of these has already been emphasized.

The late acute cases are those arising from the vesicants. The lesions do not appear until some hours after exposure, and it is therefore difficult to estimate how soon after an attack or in what numbers they will arrive at medical units. In the early stages of the lesions these casualties may be evacuated as walking or sitting cases. It is necessary to rid such cases of their contaminated clothing as early as possible so that they may not further infect themselves or others.

**130. Gas Centres.**—Special precautions must be taken to protect the personnel in dressing-stations from gas. This is more especially the case with mustard gas, since by incurring even minor burns an individual's sensitivity to this gas is apt to become increased. The likely sources of danger from mustard gas in a dressing-station arise from contaminated earth coming off the boots of stretcher-bearers who have traversed contaminated ground, from stretchers, and from the clothing and equipment of patients. The patient himself is never a source of danger; it is only his clothing and equipment.

In order to obviate the danger from boots, bleach trays should be placed outside the dressing-station and all persons instructed to rub their boots with dry bleach before entering, or if this procedure is not possible the case should be handed over outside. The problem of the danger from contaminated stretchers is a difficult one. Stretchers are likely to become contaminated from being placed on contaminated ground, or from the clothing and boots of contaminated patients. Stretchers used in a mustard area should, if it is at all possible, be protected by an improvised covering, as the difficulties of decontaminating the present type of stretcher are very great.

The clothing of patients may be dangerous when contaminated by either non-persistent or persistent gas. In the early stages of gassing by non-persistent gas, such as phosgene, where there may be practically no clinical signs, it may be necessary to resort to smelling the clothing in order to come to some decision as to whether or not the patient has been exposed to a significant dose of the gas. If this is done, care

must be exercised, and only a limited number of cases dealt with by any one individual.

If there is any possibility of the atmosphere in a dressing-station becoming contaminated with either non-persistent or persistent gas, the respirator must be worn. Frequent exposures to low concentrations of chemical warfare gases are likely to cause casualties, more especially with those gases which have a cumulative action, such as chloropicrin and mustard gas. In confined spaces, such as dressing-stations in the forward areas, the atmosphere is inclined to become heavy and the sense of smell dulled. Personnel working in such places may, in consequence, cease to appreciate after a time the presence of poisonous gas. This point should be borne in mind, and if necessary special precautions as regards wearing the respirator enforced. To obviate the wearing of the respirator in a dressing-station, the atmosphere may be kept fresh by pumping in air through a battery of containers.

Personnel may also be affected from the handling of clothing, etc., contaminated by mustard gas. It is necessary, therefore, to protect the hands. This is best effected by means of oilskin gloves. Thin rubber gloves, such as surgeons wear, should only be used in case of emergency, as they do not give a great deal of protection from liquid mustard gas. Should gloves not be available, then any improvised covering should be used, or the hands rubbed with bleach, either in the dry form or mixed into a paste with water or vaseline, which should not be applied for longer than is necessary, as prolonged and frequent applications of bleach will irritate the skin. It may be necessary to protect the clothing; this is best done by an apron, preferably of oilskin material. For the personnel employed in the sorting and handling of heavily contaminated clothing, equipment, etc., the complete protective suit should be worn.

The dressing-station itself should, if possible, be specially constructed for receiving gas casualties. The entrance should have an air-lock sufficiently long to take a stretcher case, and there should be two or more compartments, as follows:—one compartment where contaminated clothing, etc. may be removed, a second where dressings may be carried out, and a third where helpless patients who, for example, are vomiting may safely remain without a respirator.

131. When gas casualties are numerous and the military situation permits (*e.g.* in position warfare), it may be advisable to tell off certain units, *e.g.* field Ambulances or casualty clearing stations as gas centres, and to equip and organize them for this special purpose. Normally, however, all forward



medical units should be prepared to receive and treat any kind of casualty.

132. If such special centres are established it is advisable to locate them as far forward as possible, so as to avoid unnecessary movement of patients suffering from oedema of the lungs, and for the early treatment of mustard gas cases, and also so that slight gas cases may be kept as near their units as possible. In many of these cases there is a tendency to develop neurosis in one form or another; and the further such patients are removed from their units the more difficult will it be to get them back.

133. Such centres should be so organized as to facilitate rapid sorting and treatment. Accommodation should consist of a receiving room, a place for contaminated clothing and equipment, a lavage room for eye cases, baths, and wards for serious and slight cases. The wards for the serious cases should be equipped with apparatus for continuous administration of oxygen either by the Haldane apparatus, or, if this is not available, by some such methods as the following :—

A lead from the oxygen cylinder fitted with a fine adjustment valve is taken to an oil drum, which acts as a pressure reducing chamber, and from this several rubber leads branch off to nozzles which can be taken to the bedside, each being controlled by a stop-cock. This method may be developed by distributing the oxygen through lead piping, having a nozzle over each bed.

Another device enables patients already gassed to have oxygen continuously even though a wave of gas reaches as far back as the gas centre. At the bottom or top of respirator containers nozzles are fitted. These communicate by means of rubber connections with the oxygen delivery pipes. Thus, when the facepiece is adjusted the patient can obtain oxygen through his mask.

134. Though it is unnecessary to establish special hospitals at the base for the treatment of gas cases, it may be advisable to allot certain wards for research on new forms of treatment, and also to arrange in convalescent depots for special instructors to train, by graduated exercises, patients who suffer from functional disorders of the heart.

135. It may also prove necessary to have available at the base teams of specially trained medical officers, nurses and orderlies to supplement the personnel in forward medical units in times of stress.

136. Further, gas cases should, when evacuated to the

United Kingdom, be sent to hospitals staffed by personnel who have experience of their complaints. In the Great War, through lack of such experience, patients were frequently retained in hospital for unnecessarily long periods to the detriment of themselves and the service.

**137. Estimation of Casualties.**—Experience does not allow of any close estimation of the number of casualties likely to arise in the future from an intensive use of chemical weapons, but it may be stated that with highly disciplined troops, who have been thoroughly trained and accustomed to the use of the respirator, the percentage of casualties from lung irritants and toxic smokes should be low. Such casualties would chiefly arise as a result of a surprise and concentrated attack.

**138.** In the case of mustard gas the number of casualties must to a great extent depend upon whether the military situation demands traversing, or holding for a time a contaminated area. Should such a situation arise medical units must be prepared to deal with much larger numbers of casualties than those allowed for when only weapons other than chemical are used.

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## CHAPTER X

### GAS CASUALTIES ON BOARD SHIP

**139. First-aid Treatment and Disposal of Casualties on board Ship.**—The medical officer will be called upon to deal with casualties arising from persistent and non-persistent gases. He will have made himself acquainted with their mode of action, the symptoms and pathology produced by them and their treatment, and be in a position to determine the kind of gas producing the casualties at the time. It is not anticipated that casualties caused by lung irritants, except nitrous fumes, will be met with at sea, and the probability of any casualties arising from the use of lachrymators and sensory irritants is a remote one. Should such gases, however, be used, the immediate wearing of the respirator will afford complete protection. It is extremely important that the medical officer should bear in mind the possibility of some of the personnel seeking medical advice on flimsy grounds of being gassed. It is of the greatest importance that every man in a ship should remain at his



post, and it is incumbent upon the medical officer to impress upon all personnel in a ship that the inhalation of a moderate amount of sensory irritants, or the irritant effects of a lachrymator, present no danger, and cause nothing more than a temporary discomfort. There should not be any cases calling for first-aid treatment, and there will be no necessity for any of the personnel to leave their post. He will, however, have to exercise the greatest care in the differentiation of such cases from those caused by the inhalation of nitrous fumes, which are of a severe and dangerous nature. These cases will be dealt with later.

140. The most important persistent gas is mustard gas ; but even although it may have been freely used by an enemy, the effect of direct contamination of personnel can be minimized greatly by the following first-aid treatment. Immediately liquid mustard is seen on the skin an ointment made of 2 parts bleach and 1 part vaseline should be rubbed in. The ointment should be wiped off after a few minutes. If the mustard gas contamination be recognized and the ointment used without delay, serious burning of the skin should not occur. It may, therefore, be desirable to distribute throughout the ship supplies of bleach ointment, so that persons who are contaminated may cleanse themselves from mustard gas at once and remain at their posts of duty. Contamination of the clothing must be dealt with as circumstances permit.

Care should be taken that wounded, or the stretcher-bearers, are not admitted to the medical distributing station with mustard gas contamination of their clothing or stretchers. Those in urgent need of surgical treatment must have their clothing rapidly removed outside the station before being admitted to hospital. These matters are discussed at greater length in para. 130.

### **Toxic Gases not used as Offensive Weapons.**

141. Of all the toxic gases not used as offensive weapons, the most important and those most likely to be met with are :—

- (a) Nitrous fumes.
- (b) Carbon monoxide.
- (c) Arseniuretted hydrogen.
- (d) Chlorine.
- (e) Ammonia.

142. (a) **Nitrous Fumes.**—These are given off when detonation is incomplete, or from burning cordite. The more imperfect the combustion, the greater will be the amount of nitrous

gases produced. The period of exposure for the production of symptoms varies with the percentage of the nitrous gases present, the more prolonged the exposure, the more rapid and serious will the results be. In some cases a very short exposure may produce serious results. The initial symptoms at the time of exposure are slight irritation in the nose and throat, a feeling of constriction and perhaps pain in the chest, some irritating cough, accompanied by headache, smarting of the eyes and perhaps a slight attack of vomiting. These initial symptoms are merely transitory, and then follows a latent period during which the patient feels quite well again. This latent period or time of delayed action varies from two to twenty-nine hours, taking the extreme cases. The greatest number of cases occur, however, between the tenth and twentieth hour after exposure to the fumes. Because of the slight initial effects, these fumes are particularly dangerous, as men may fail to realize the danger. Once the latent period is over, the symptoms and signs of acute pulmonary congestion and œdema, with its accompanying cyanosis, appear rapidly. This condition may be ushered in by a stage of irritation and spasm, just as in the cases of chlorine poisoning, and is followed by a state of acute pulmonary congestion and œdema, which in fatal cases may occur with almost explosive violence, frothy blood-stained fluid pouring from the mouth and nostrils, while the patient tosses wildly about in bed, vainly struggling for breath, and acutely conscious of his sufferings all the time.

With regard to treatment, in the case of men who have undoubtedly been exposed to nitrous fumes, from whatever cause, it is most important to take them off duty for the next 48 hours, and keep them under observation and at rest for the period of the delayed action, so that the cases as they arise may receive the immediate medical attention that is so essential for their cure.

To relieve spasm before secretion is great, and when it has begun, an emetic such as zinc sulph. gr. xxx is very useful in cases where vomiting has not occurred, as the vomiting is accompanied by a great amount of frothy fluid.

143. Oxygen administration and venesection are extremely important. These two forms of treatment will be found detailed at paras. 59 and 60 of this Manual. The use of cardiac stimulants and expectorants will be helpful as the occasion arises. The same precaution of keeping the patient at rest after being gassed, as pointed out in the case of phosgene earlier in this manual, is imperative.

144. Men should be warned that air which has even the slightest odour of nitrous gases is dangerous, and that, although



it may only give rise to trivial respiratory discomfort at the time, serious results are likely to follow later. When explosions, or fire involving explosives, occur on board a ship, the area involved should be a prohibited one unless masks are worn. The service respirator protects against nitrous fumes for a short time, but the compartment should be vacated and ventilated as quickly as possible.

145. (b) **Carbon Monoxide.**—This gas will often be found associated with nitrous fumes. Its mode of action, pathology, symptoms and treatment will be found at para. 92 *et seq.* of this manual. Special attention must be drawn to the treatment by breathing air mixed with carbon dioxide to the extent of 5 to 7 per cent in view of the fact that cylinders of carbon dioxide are always available in most ships. As the service respirator does not afford protection against carbon monoxide, the service shallow-water diving dress, which is a self-contained oxygen apparatus, can be used.

146. (c) **Arseniuretted Hydrogen.**—Cases of poisoning by this gas occurred during the war in certain submarines, owing to its production by the use of impure materials in the batteries.

This gas is typically cumulative in action. Exposure of animals for about six minutes to 1 part of this gas in 1,000 may cause death in 24 hours. With lower concentrations the effect is nearly proportional to the product of the concentration of the gas and the duration of the exposure, a concentration of 1 part of the gas in 100,000 being fatal after about twelve hours' exposure.

147. *Mode of Action.*—The gas acts upon the red blood corpuscles, causing intense hæmolysis, and this effect seems to be the main cause of the symptoms. In slight cases there is only jaundice and anæmia, associated with a feeling of malaise, nausea, and pains about the body. In more severe cases there is hæmoglobinuria as well as jaundice and profound secondary anæmia. In bad cases vomiting sets in in an hour or two, with intense pain in the back, weakness, fainting and collapse; hæmaturia is intense, and acute nephritis occurs with casts in the urine. In fatal cases there may be total suppression of the urine. Death seems to be due either to the nephritis or to the want of oxygen caused by the destruction of the red blood corpuscles and loss of hæmoglobin. The nephritis appears to be largely dependent on the accumulation of hæmoglobin or its derivatives in the kidney. Peripheral neuritis is a not uncommon symptom in the latter stages of non-fatal cases.

148. In the cases which occurred in submarines, the main symptoms were: vomiting—a constant and troublesome symptom often associated with burning and griping abdominal pain—dyspnoea on exertion, anæmia, jaundice, hæmoglobinuria, and albuminuria, headache and slight œdema of the face and eyelids. Many of the cases gave evidence of a slight degree of neuritis in the shape of tingling and “pins and needles” in the hands and feet. The anæmia was occasionally severe, for some of the cases admitted to hospital showed only two million red corpuscles to the cubic millimetre of blood and a hæmoglobin percentage of only half the normal amount. Hæmoglobinuria ceased within three days of leaving the submarine, though slight albuminuria continued in an intermittent fashion for some time longer. The jaundice usually disappeared within a week or ten days. The symptoms of neuritis took two or three weeks to pass off, and a considerably longer time was required before the blood resumed its normal character, though the cases had all recovered completely at the end of six weeks.

149. (*d*) **Chlorine**.—This gas may be encountered, particularly in submarines, through the accidental access of sea water to the batteries when chlorine is evolved by electrolysis. The respirator gives complete protection.

150. (*e*) **Ammonia**.—This gas may occasionally be met with in certain ships in which it is utilized for refrigerating and cooling purposes. It is carried in the liquid form in cylinders. The casualties likely to result from the accidental liberation of this gas will be from inhalation affecting the respiratory passages, and from its action on the eyes and skin. The upper air passages and bronchioles are chiefly affected, these parts exhibiting well-marked signs of irritation and inflammation with formation of tenacious mucus, suggestive of an acute bronchitis. Coughing, burning in the throat and vomiting are frequently present. The severity of the symptoms will depend upon the concentration of the gas. As regards skin effects, these again will depend upon the concentration, varying from marked and immediate skin irritation in high concentrations to a chronic dermatitis in the case of a long-continued exposure to a low concentration. As regards the eyes, well-marked lachrymation is produced, and in the case of long-continued exposure to low concentrations, a chronic conjunctivitis is likely to develop.

151. *Treatment*.—In case of casualties resulting from inhalation, immediate removal from the atmosphere containing the fumes is the first essential. A cloth dipped in vinegar and



applied to the mouth and nose will afford relief. Irritation to the eyes should be treated by bathing with boric lotion. Skin trouble should be treated on the ordinary lines.

152. The service container will protect against ammonia in a concentration of 1 in 50 by volume (which is, of course, very high) for  $6\frac{1}{2}$  minutes, and for longer periods against lower concentrations. As ammonia is very soluble in water some measure of protection is afforded by the application of a cloth soaked in water and applied to the nose and mouth. For the same reason, if water is sprayed on a leak, the concentration will be greatly reduced.

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# APPENDIX I LETHAL AND INCAPACITATING CONCENTRATIONS FOR AN UNPROTECTED MAN

	Effective concentration ( <i>i.e.</i> concentration which will incapacitate) for unprotected men for exposures of :—		Lethal concentration for unprotected men for exposure of :—		Concentration which can be breathed in— definitely without damage.
	2 minutes	10 minutes	2 minutes	10 minutes	
Diphenyl chloroarsine ...	1 : 7·5 million	1 : 75 million	1 : 8,000	1 : 40,000	1 : 1,000 million
Diphenylamine chloroarsine ...	1 : 7·5 "	1 : 75 "	1 : 8,000	1 : 40,000	1 : 1,000 "
Diphenyl cyanoarsine...	1 : 17 "	1 : 170 "	1 : 16,000	1 : 80,000	1 : 2,500 "
Ethyl iodoacetate ...	1 : 5 "	1 : 10 "	(1)	—	1 : 100 "
Chlorine ...	1 : 10,000 "	1 : 40,000 "	1 : 2,000*	1 : 10,000*	1 : 175,000 "
Phosgene ...	1 : 70,000	(2)	1 : 10,000	1 : 50,000	1 : 1 million
Chloropicrin ...	1 : 100,000	1 : 200,000	1 : 4,000	1 : 20,000	1 : 1 "
Chloroacetophenone ...	1 : 5 million	1 : 100 million	—	—	1 : 250 million

\* *Provisional figures.*

(1) In practice a lethal concentration can hardly be obtained in the field.

(2) A concentration which is just insufficient to cause immediate coughing or lachrymation can usually be tolerated, since the initial irritation tends to subside. Such an atmosphere is extremely dangerous as continued breathing of it may in the end cause a fatal result.

NOTE.—This table may be accepted as a rough guide from the defensive aspect alone ; that is, the concentrations and times stated may, with every condition in their favour, produce the result stated.



## APPENDIX II

## HALDANE OXYGEN APPARATUS

In using oxygen for medical purposes it is very necessary to have on the cylinder (i) a pressure gauge to tell how much oxygen is present in the cylinder, and (ii) a reducing valve, so constructed as to reduce the pressure to manageable proportions, which will allow of its remaining constant till the cylinder becomes almost empty.

In the Haldane apparatus there is also a regulating valve which is graduated to deliver from 1 to 10 litres a minute. From this a flexible tube leads to the mask. A small bag is attached either to the mask itself, or close to the regulating valve to act as a reservoir for the oxygen. From the reservoir oxygen enters the mask through a mica non-return valve. In front of the mask there is an aperture in which a rubber flap hangs. The object of this is to allow of the passage of the inspired and expired air against a very slight resistance. Elastic head harness is attached to the mask to hold it in position on the face.

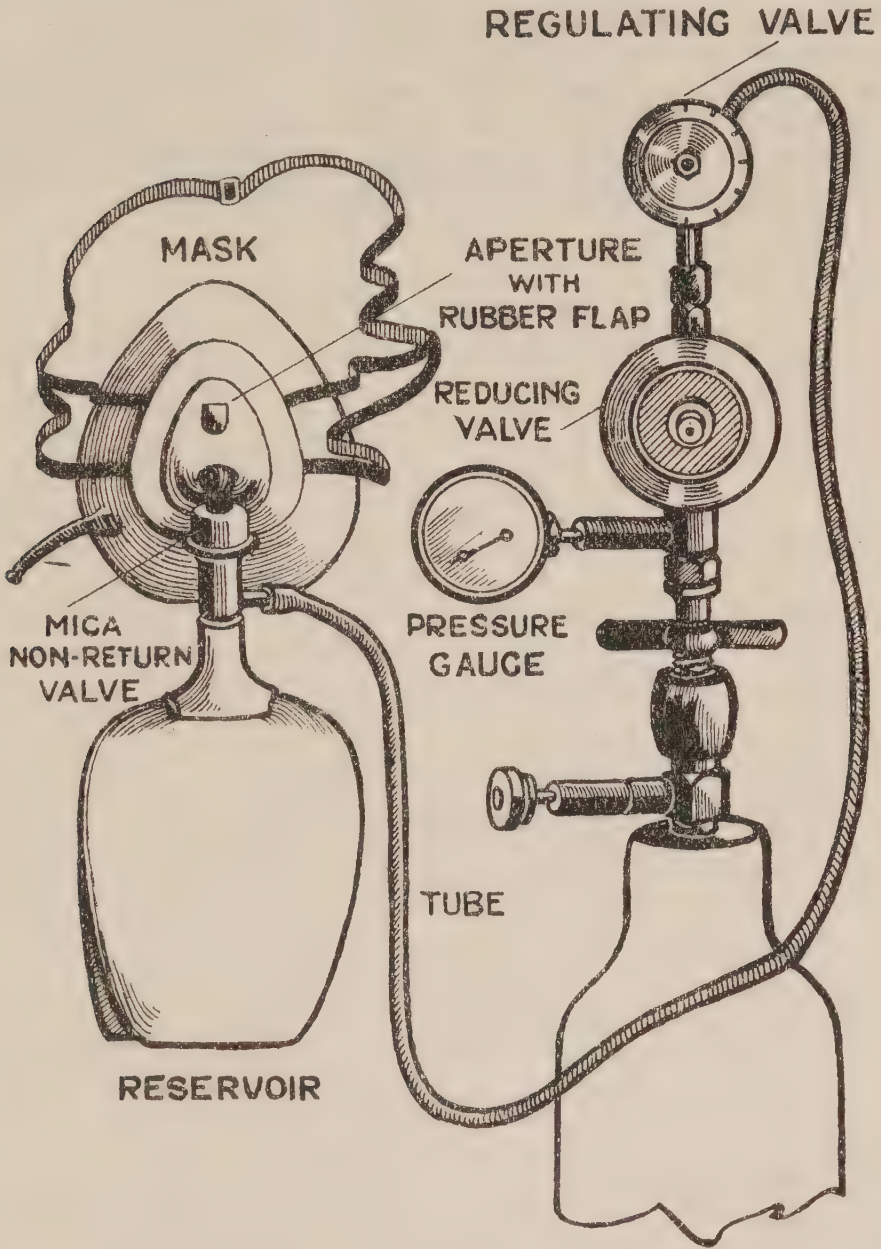
The reducing valve is set by the makers and should not be interfered with.

To start the oxygen the main valve of the cylinder is opened, and the regulating valve set to the required delivery. The reservoir bag then fills up and during inspiration oxygen is drawn from this into the mask through the mica valve. The oxygen entering the mask is diluted with air drawn through the rubber-flap aperture. During expiration the non-return valve closes and the expired air escapes through the rubber-flap aperture in the front of the mask. *See figure.*

The mask fits the face lightly, though fairly closely. The drawbacks of the mask are obvious. A casualty suffering from embarrassed breathing will not relish the idea of having anything put over his nose and mouth which may still further hinder his breathing. Very often the patient's objection to the mask can be overcome by gaining his confidence. It is worth while spending a few minutes trying to do this, holding the mask a short distance away from the face and gradually bringing it up into position. In this connection there is a practical point worth noting. When the mask is first applied the patient is apt to take a very deep breath. If he encounters any undue resistance his discomfort will be increased, and he will probably refuse to give the mask a further trial. To overcome this the reserve bag should always be completely filled with oxygen before the mask is applied.

The initial gasp will give relief and he will be encouraged to proceed.

The minimal current should be used ; the actual amount depends entirely on the individual case, and must be gauged by



the disappearance of the cyanosis and the improvement of the pulse. The flow may be anything from 2 to 10 litres a minute and may require to be kept going for anything up to 7 days. When oxygen is given for prolonged periods an intermission of five minutes every half hour is recommended.



The general condition of the patient and the colour of the lips are the best guide to the length of time and the amount of the flow.

Haldane's apparatus has the advantage that the oxygen flow can be readily gauged and regulated, and that, in addition, what flows during expiration is not completely wasted. The importance of cutting down waste is vital where, as in the great majority of cases, continuous administration of oxygen over many hours, or even several days is needed. The difficulties in transport or handling of heavy oxygen cylinders ought to be reduced to the minimum possible.

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